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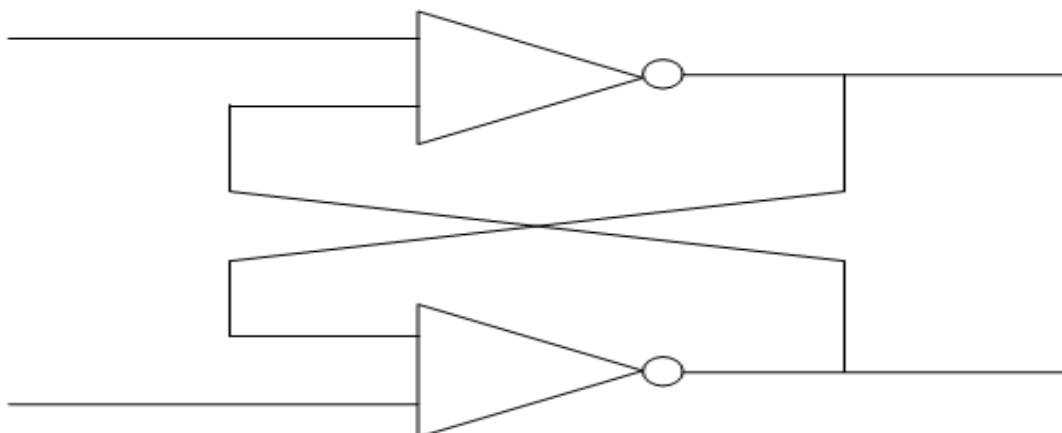
### FLIP FLOP MODEL IN BASIC MEDICAL SCIENCES

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Only change is eternal. We have learned from homeostasis that internal environment must be kept constant whatever changes take place in external environment. To keep internal environment constant we know we have established models feedback mechanisms i.e. positive feedback and negative feedback mechanisms. But certain changes are so sudden i.e. from wakefulness to sleep or sleep to wakefulness; such changes are better explain by flip flop model. Flip flop circuit model is often used in computer and electronics and called bistable an electronic circuit that can assume either of two stable states by the application of a suitable pulse. So flip-flop circuit is a loops having two stable conditions, each one corresponding to one of two alternative input signals. This two component circuit model is, however, evocative of Walter Cannon's original formulation of the homeostasis concept which referred to the balance of two opposing factors. The flip flop circuit analogy is taken from electronic engineering and provide framework for functioning of two mutually influential circuits in brain i.e. awakening circuit and sleep inducing circuit. Though it appear to be a form of positive feedback loop but it has a switch like component which decide either of two bistable circuit. It can only exist in one of two states interdependent but quiet opposite to each other. It is found in nature where tight control is needed. The most important feature of this Positive feedback circuit is that they help to make the rapid switch from one state to another state. We have just began to identify these switches which can on and off, so that only the correct state of two state is assumed. Such switches in central nervous gramsystem are frequently a center or clusters of neurons secreting specific neurotransmitters which decides on-off states in either side.

#### Diagram showing analogy of flip flop circuit in electronics



In electronics memory i.e., a flip-flop or latch is a circuit that has two stable states and can be used to store state information. The circuit can be made to change state by signals applied to one or more control inputs and will have one or two outputs. It is the basic storage element in sequential logic. Flip-flops and latches are a fundamental building block of digital electronics systems used in computers, communications, and many other types of systems. The word *latch* is mainly used for storage elements, while clocked devices are described as *flip-flops*.

In reference to neuro physiology, during sleep and wake states, the most of the cortical columns are in their respective sleep-like and wake-like states, suggesting synchrony of state between columns. Cortical columns are connected to subcortical sleep regulatory circuits. Von Economo and Moruzzi, work concluded that cortical arousal is regulated by subcortical regions. Ascending reticular activating system [ARAS] originated from brain by two major branches of ARAS. [1] Cholinergic cell groups in the upper pons input into thalamus and densely innervate cortex. This system is responsible for either wakefulness and REM sleep. [2] The other second branch originates from lower pons and locus coeruleus (norepinephrine), dorsal and medial raphe (serotonin), and tuberomammillary cells (histamine) to innervate lateral hypothalamic region, basal forebrain and cortex. End target neuron of this system is cholinergic or GABAergic (GABA) –mediated neurons. This branch fire maximally during wakefulness and slowing or absent during NREM sleep. Discovery of orexin (also called hypocretin) from the lateral hypothalamus through more light in understanding how sleep is initiated, maintained and terminated. It is postulated that there is a switch i.e. being in the ventrolateral preoptic area (VLPO) of hypothalamus. VLPO area maximally active during sleep and major output to monoaminergic neurons of brain stem and basal region and neurotransmitters are inhibitory (ie, galanin and GABA). Saper and colleagues postulated VLPO function as an “off switch” to inhibit arousal. Lesion in this region reduced sleep. VLPO not only inhibits wakefulness but also inhibited by wakefulness. This reciprocal relationship between the VLPO and the ARAS is the functioning of a “flip flop circuit.” These studies suggest that mutually inhibitory wake and sleep promoting circuits (“flip-flop switches”) are an evolutionarily conserved mechanism to promote fast and complete state switching between wake and sleep. Similarly Hunger and fed states are also an example of a flip-flop circuit via a synaptic AMPK-dependent positive feedback circuit pathway for physiologic regulation. Here “set”: ghrelin and “reset”: leptin hormones or neurotransmitters separately maintain flip flop circuit. In this circuit, though, we add the provision that these signals operate as discrete Boolean logical operations due to an AMPK-dependent positive feedback loop. For physiological state constructed from bistable synapses that are flipped between sustained high and low activity states by transient exposure to hormones associated with energy deficit and surfeit, respectively.

Flip flop model is likely to be useful to explain some regulatory mechanisms i.e. circadian and diurnal rhythms, regulation of heart beat, cardiac arrhythmias i.e. circus phenomenon, neural and chemical regulation of respiration. The model already helped to explain moment of phospholipid particles on cell membrane and many intracellular and intranuclear and overment genetic molecules [molecular switches] some biochemical mechanisms i.e. lipid signaling.

**References:**

- 1] Christopher J. Davis, PhD, James M. Krueger, PhD\*, Sleep and Cytokines.  
A book biology of sleep-sleep medicine clinics. sept 2012, vol.7 - no. 3 ,published by Elsevier publication
- 2] Michael Perlis, PhD<sup>a\*</sup>, Wil Pigeon, PhD<sup>b</sup>, Phil Gehrman, PhD<sup>a</sup>, Jim Findley, PhD<sup>a</sup>, Neurobiological Mechanisms in Chronic Insomnia, Sean Drummond, PhD<sup>c</sup>, A book biology of sleep-sleep medicine
- 3] Saper CB, Chou TC, Scammell TE. The sleep switch: hypothalamic control of sleep and wakefulness. Trends Neurosci 2001; 24: 726-31.
- 4] [Yunlei Yang](#), [Deniz Atasoy](#), [Helen H. Su](#), and [Scott M. Sternson](#)\* Hunger states switch a flip-flop memory circuit via a synaptic AMPK-dependent positive feedback loop, CELL. 2011 September 16; 146(6): 992–1003. PMCID: PMC3209501

**(1) MEDITATION IS NOT MERE RELAXATION': COMPARISON OF EFFECTS OF MEDITATION AND RELAXATION ON BASIC CARDIORESPIRATORY PARAMETERS.**

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**Abstract****Introduction:**

In today's hectic routine we are being subjected to increasing levels of stress, which has become a part and parcel of our life and just can't be avoided. Mankind is thus increasingly on the lookout for various lifestyle modifications. Amongst these modifications, Meditation is proving out to be one of the most universally feasible solution to this modern day stressful condition.

**Objective:**

Widely proclaimed therapeutic benefits of meditation have motivated us to conduct the present study in order to evaluate the physiological response patterns obtained by meditation on basic cardio respiratory parameters like pulse rate and respiratory rate.

**Method:**

The study was conducted on 30 Meditators and 30 Non-meditators. The Non-meditators were used as controls and were made to relax in the lying position. Pulse rate and respiratory rate were taken in three phases in both the groups, that is 1) before 2) during and 3) after meditation or relaxation as per the respective group.

**Results:**

Analysis of the study revealed a significant fall in respiratory rate as well as pulse rate in the meditators during phase 2 and phase 3. But similar effects were not observed in the Non-meditator group in any of the phases of relaxation.

**Conclusion:**

Meditation causes decrease in both respiratory rate as well as pulse rate and thereby the heart rate.

**Key words**

Pulse rate, Respiratory rate, meditation, relaxation

**Introduction**

Considering today's stressful and modern-day hectic schedules Humanity is increasingly turning towards various lifestyle modifications. Unable to locate stability and serenity in the outside world people have directed their gaze inwards in a bid to attain peace of mind. Meditation is the first mind body intervention to be widely adopted in mainstream health care in the western society and around the world. Varied positive physiological, psychological and spiritual benefits are known to be achieved through meditation. Meditation since generations has helped most people feel less anxious, and results in a state of relaxation, by being a source of personal insight and self-understanding. These subjective experiences may have relevance from a clinical or research perspective. Transcendental meditation, Sahajayoga, Vipassana and Mindfulness meditation (1) are some of the more established methods of meditation.

Generally the entire process of meditation entails the following stages: concentration (dharana), absorption (jhana) and one pointedness (samadhi) often followed by the stage of knowledge (nana) (2).

Meditation is a wakeful hypometabolic state which is accompanied by a decreased breathing pattern, decreased heart rate and decreased blood pressure. There is also a marked decrease in level of O<sub>2</sub> utilization and CO<sub>2</sub> elimination by the muscles (3,4,5,6,7). This reduced metabolic status is brought about at the cellular level as an effect of meditation and not from a forced reduction of breathing (4). Meditation also due to its effect on the autonomic nervous system has been shown to affect heart rate and cutaneous peripheral vascular resistance (6). Taking into account the above benefits the present study was thus conducted to find out the effect of meditation on biophysical parameters like pulse rate and respiratory rate.

## Materials and Methods

30 meditators and 30 non-meditators were included in the present study. The study was conducted at Grant Govt. Medical College and J. J. Hospital, Mumbai, after obtaining approval from the institutional ethical committee. The selection criteria of the subjects who were in the age group of 27 to 65 years were as follows.

- 1) Strict vegetarian diet.
- 2) No smoking habit.
- 3) No history of alcohol intake.
- 4) No past history of hypertension.
- 5) Systolic Blood pressure not more than 130 mm Hg and diastolic Blood pressure not more than 90 mm Hg.
- 6) Not a known case of hyperglycemia or hyperlipidemia.

'Sadhaks' from Mumbai based Prajapati Brahmakumari Ashram who were practicing regular meditation for a minimum period of 6 months prior to the conduction of study comprised the subjects of Group I (meditators).

Volunteers randomly chosen from the normal healthy population who never practiced any kind of meditation formed the subjects of Group II (non-meditators) who were used as controls.

The procedure for the study was carried out during the morning hours in the following manner.

Group I subjects were asked to perform 30 minutes of concentrative meditation.

Group II subjects were asked to just relax by lying supine for 30 minutes.

Respiratory Rate and Pulse rate were taken in both the groups in three different phases, that is 1) prior to 2) during and 3) after their respective procedures. Respiratory rate was counted by the standard routine procedure of observing the movements of the chest wall of the subjects for one complete minute. The pulse rate was counted in the Radial artery of the Right forearm by palpatory method for one complete minute. A stop watch calibrated to 1/100<sup>th</sup> of a second was used to measure the time for both the parameters. The observations thus obtained were noted down and used for subsequent statistical analysis.

## Statistical analysis

The statistical analysis of the data obtained for the meditator and non meditator groups were done using paired t test and p value of less than 0.05 was considered significant and those less than 0.001 was considered highly significant. The results obtained on analysis were presented as Mean  $\pm$  Standard Deviation for each of the parameters.

## Observation and Results

The meditator group showed highly significant reduction ( $p < 0.001$ ) in respiratory rate as well as pulse rate on comparison of phase 1 with phase 3. In group II subjects undergoing relaxation significant reduction ( $p < 0.05$ ) in pulse rate was noted while comparing the pulse before and after relaxation. But on comparison of respiratory rate no such reduction was found between phase 1 and phase 3 of relaxation.



**Table 1** Showing changes in Respiratory rate and pulse rate during phase 1, 2 and 3 in meditators and non-meditators

	<b>Group I Meditators (undergoing Meditation)</b>		<b>Group II Non-Meditators (undergoing Relaxation)</b>	
	Resp. Rate	Pulse rate	Resp. Rate	Pulse rate
Phase 1 Before meditation/Relaxation	11.60±1.50	78.60±5.85	11.90±1.57	77.30±6.35
Phase 2 during meditation/Relaxation	8.80±2.99	71.50±6.64	11.70±1.46	76.10±7.00
Phase 3 after meditation/Relaxation	9.20±2.99	70.70±5.47	11.70±1.46	76.10±7.00
p value (Significance)	p* < 0.001 (HS)	p* < 0.001 (HS)	p* > 0.05 (NS)	p* < 0.05 (S)

NS-not significant, S-significant, HS-Highly significant.

\*comparison of 'before' with 'after' the respective procedure.

Significant difference, that is lower pulse rate as well as respiratory rate were observed in the meditators in comparison to the group undergoing relaxation during phase 2 of the respective procedures. Phase 3, that is results seen after the respective procedures too showed significant lower values of pulse rate as well as respiratory rate in the meditator group in comparison to group II.

**Table 2** Showing the Comparative effects of meditation and relaxation on respiratory rate and pulse rate in the three different phases.

	<b>Respiratory Rate</b>		<b>Pulse rate</b>	
	<b>Group I (meditation)</b>	<b>Group II (relaxation)</b>	<b>Group I (meditation)</b>	<b>Group II (relaxation)</b>
Phase 1 Before meditation/Relaxation	11.60±1.50	11.90±1.57	78.60±5.85	77.30±6.35
Phase 2 during meditation/Relaxation	8.80±2.99	11.70±1.46	71.50±6.64	76.10±7.00
Phase 3 after meditation/Relaxation	9.20±2.99	11.70±1.46	70.70±5.47	76.10±7.00
value (Significance)	p* > 0.05 (NS) p** < 0.05 (S) p*** < 0.05 (S)		p* > 0.05 (NS) p** < 0.05 (S) p*** < 0.05 (S)	

NS-not significant, S-significant,

\*comparison of group I with group II during phase1.

\*\*comparison of group I with group II during phase2.

\*\*\*comparison of group I with group II during phase3.

## Discussion

Various biophysical parameters are determinants of the status of the health of an individual and are the subjects of interest for clinical research and investigation. Meditation shows to have a strong evidence to modify, regulate and improve many of these biophysical parameters. Out of these parameters respiratory rate currently seems to be the least studied parameter amongst meditators. On the contrary many studies have been done on pulse rate and heart rate to study cardio respiratory status in meditators and other individuals. Findings showing the beneficial effect of meditation by reducing or normalizing the pulse have been replicated in many studies.

R. Schneider in 2006(8) has shown reduction of pulse rate in regular practitioners of meditation.

Khare et al in 2000(11) in a similar study like ours in which pulse rate was measured before, during and after meditation had found pulse rate reduction in the 'during' and 'after' phases of meditation.

S. Terathongkum in 2004(12) too had shown a significant decrease in heart rate in meditators.

Similarly S. Maini et al in 2011(13) had studied the effect of rajyoga meditation on heart rate, blood pressure and ECG and had found reduction in heart rate in the meditator group.

Vyas et al in 2002(9) in their study had found reduction in pulse rate in both long term and short term meditators, thereby showing the beneficial effect of even short term practice of meditation.

Our study showed statistically significant reduction in pulse rate in the meditator group during and after meditation. These findings were quite consistent with most of the research works conducted by others.

Increased Sympathetic activity is known to increase heart rate and pulse whereas increased parasympathetic activity is known to decrease the heart rate and pulse. Meditation is known to have an autonomic regulating action by activating the parasympathetic nervous system.

The parasympathetic-limbic activation achieved through meditation relaxes us by balancing the two opposing sympathetic functions. (16, 17) This relaxed state in meditation is accompanied by alterations in certain physiological parameters one of which is decrease in pulse rate. (15, 18)

Out of the different physiological parameters the fewer studies done on respiratory rate in the current scenario are worth mentioning.

Khare et al in 2000(11) had found significant fall in respiratory rate in the 'during' and 'after' phases of meditation in their study in which they had used grass model 5 polygraph.

Dudhmal et al in 2004(10) had demonstrated significant reduction in the respiratory rate on comparing 'before' and 'after' values obtained after 10 days of vipassana meditation.

In our present study we found a significantly reduced respiratory rate in the 'during' and 'after' phases of meditation. These findings were quite comparable to the study done by khare et al in 2000.

As mentioned before, meditation is known to cause a hypometabolic effect on the body which leads to a decreased oxygen consumption and also decreased elimination of carbon dioxide. This has a direct effect on reducing the respiratory rate. (3, 4, 14)

Another explanation could perhaps be also cited to the fact that Meditation is known to have an autonomic regulating action by balancing the parasympathetic and sympathetic nervous system in conjunction with the limbic system. This relaxed state obtained due to meditation is accompanied by a decrease in the respiratory rate, which is one of the various physiological parameters that have been shown to change after meditation is introduced to a person. (15, 18)

### **Conclusion and summary**

Thus, we conclude from the present study that meditation causes decrease in both respiratory rate as well as pulse rate and there by the heart rate. This thereby explains that the relaxed state which one experiences subjectively in meditation is in fact accompanied by alterations in various physiological parameters that have been traditionally related to stress. Meditation is thus more than mere relaxation and thereby an introduction of meditation in our day to day routine should definitely be encouraged to cope up with today's demanding lifestyle and daily stress.

### **References**

1. R. Manocha. Why meditation? *Australian Family Physician J* Dec 2000; 29:12.
2. Ken Wilber, Jack Engler, Daniel. P. Brown et al. Transformations In consciousness : Conventional & Contemplative perspectives on development. Ch 1:32.
3. Wallace. R. K. Physiological Effects of Transcendental meditation. *Signs* 1970; 167:1751-1754.
4. Wallace. R. K., Benson H, Wilson A. A wakeful hypometabolic Physiologic State. *Am J Physiol* 1971; 221:795-799.
5. Delmonte. M. M. Physiologic responses during meditation and rest. *J Biofeedback and Self Regulation* June 1984;9 (2):181-200.
6. Shirley Telles, Nagarathana R, Nagendra. H. R. Autonomic changes during "OM" Meditation. *Ind. J Physiol & Pharmacol* Oct 1995; 39 (4):418-420.
7. Vyas R, Dixit N. Effect of meditation on respiratory system, cardiovascular system and lipid profile. *Ind. J Physiol & Pharmacol* Oct 2002; 46(4):487-491.
8. Noel Bairey Merz, Robert Schneider. Meditation may improve cardiac risk factors in patients with coronary artery disease. *JAMA* 2006; 166 (11):1218-1224.
9. Vyas R, Dixit N. Effect of meditation on respiratory system, cardiovascular system and lipid profile. *Ind. J Physiol & Pharmacol* Oct 2002; 46 (4):487-491.

10. Solpure AB, Dhudhmel VB, Sayeda Afroz. Effect of vipassana meditation on serum level of malondialdehyde & some cardio respiratory parameters. *Milestone* 2004;3:4.
11. Khare, Nigam. Study of electroencephalogram & cardio respiratory parameters in meditators and non-meditators. *Ind. J Physiol & Pharmacol* April 2000; 44:2.
12. S. Terathongkum. Heart rate variability, Blood pressure changes in meditators during and after meditation 2004;22(3):78-82.
13. Seema Maini, Harleen Kaur, Navneet Maini. Effect of Rajyoga meditation on heart rate blood pressure and ECG. *J Clin & Diag Res.* 2011; 5 (8):1519-1522.
14. Fenwick P.B.C. Metabolic and EEG changes during TM Meditation: An Explanation. *Biological Psychol.* June 1977; 5 (2):101-118
15. Rai, U. C., S. Setji, and S. H. Singh. "Some effects of Sahaja Yoga and its role in the prevention of stress disorders." *Journal of International Medical Sciences*1988;19: 23.
16. <http://www.haveahealthymind.com/articles/stress-response-system.html>
17. [http://www.theelementsofhealth.com/resources/articles/articles-stress/the\\_autonomic\\_nervous\\_syste.pdf](http://www.theelementsofhealth.com/resources/articles/articles-stress/the_autonomic_nervous_syste.pdf)
18. Rai, Umesh C. Medical science enlightened: new insight into vibratory awareness for holistic health care. Life Eternal Trust, 1993.

## (2) INTEGRATED TEACHING - TOOL FOR REFORMATION OF CURRICULU

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### INTEGRATED TEACHING - TOOL FOR REFORMATION OF CURRICULUM

#### Introduction

Traditional teaching learning methods follow 'Discipline Wise Model'. This leads to unnecessary repetition, disjointed approach to teaching, and confusion in student's mind due to difference of opinion leading to improper grasping of the subject. This discourages students from learning & they get disinterested in applying knowledge achieved into practice.

Hence, the integrated teaching is need of today's medical education. So we conducted the study to compare scientific method for teaching and also obtained student's perspective.

## Aims

To analyze student's feedback after introduction to new teaching-learning method, to make them learn to integrate the knowledge across the disciplines, to analyze the impact of integrated teaching over student, to improve the student's interest.

## Materials & Method

48 students of 5<sup>th</sup> semester were involved in the study. After going through the Maharashtra University syllabus of Pathology & Physiology, two overlapping topics were selected. The students were divided into two symmetrical groups (A & B). For first topic, group A was given traditional & group B was given integrated lecture. Reverse was done for second topic. Both the traditional lectures were taken with OHP whereas the integrated lectures were taken with LCD presentation. Data from Pre- & Post-tests with prevalidated MCQs & the student's feedback with prevalidated questionnaire containing both close & open ended questions were statistically analyzed using 't' test (paired, unpaired) & Z test.

## Result & Analysis

The statistical value for pre- & post-test of traditional & integrated lecture was 6.704 & 8.100 respectively. The statistical values for post-test 1 & post-test 2 of traditional & integrated lecture were found to be 0.756 & 0.332 respectively. 87.5% students thought that integrated lecture is more effective than traditional & 12.5% thought vice versa. 37 students felt that LCD is better than OHP. 20.83% students found that it is easy to write notes in traditional lectures & is less time consuming & 66.67% students felt that they were monotonous, boring & sedative. 95.83% students wanted more integrated lecture. 22 students felt that it is more effective & explanatory, supportive in 3<sup>rd</sup> year with more animations & pictorial presentation.

## Discussion

Students found the integrated learning programme to be a useful, feasible method of learning. However, the statistical comparison between the evaluation after integrated & traditional lectures had not shown significant impact of integrated lectures. But the statistics reveal certainly that students felt the need for integration.

This study was an attempt to improve the quality of medical education with the innovative curricular strategy. The lacunae of the study were the small sample size & long-term impact. The integrated teaching would surely reduce the fragmentation of medical course; prevent repetition, waste of time & rationalization of teaching resources. It will also promote the interdepartmental collaboration & students will learn to apply their knowledge to clinical practice.

## Introduction

In today's medical education methods, we learn all about human body in various departments as separate compartments related to the body. But even the human body works in a very well coordinated, integrated fashion to achieve the homeostasis. Medical education cannot be categorised at different levels. Many topics in basic sciences are taught to medical students in 'Discipline Wise Model' i.e. compartmentalization. This leads to unnecessary repetition, disjointed approach to teaching, and confusion in student's mind due to difference

of opinion leading to improper grasping of the subject. This discourages students from learning & they get disinterested in applying knowledge achieved into practice.

As the medical education is related with the community services, we need to teach our students to correlate the various subjects to create good doctors. Teaching the same topic in sequence from the different faculty members will better assimilate the knowledge. This will impart the basic knowledge of the topic for better understanding of the various aspects of the diseases, its progress & management.

Traditional teaching-learning methods are mainly didactic & passive. Hence, the integrated teaching is need of today's medical education to create better doctors in society who will provide good health care services to community needs. So we wanted to compare scientific methods for teaching and also obtained students' perspective.

### **Aims & Objectives**

1. To analyze student's feedback after introduction to new teaching-learning method.
2. To make them learn to integrate the knowledge across the disciplines.
3. To analyze the impact of integrated teaching over student.
4. To improve the student's interest.

### **Abbreviations used:**

OHP – Overhead Projector  
LCD – Multimedia Projector  
IL – Integrated Lecture  
TL – Traditional Lecture

### **Materials & Method**

This cross sectional study was conducted in department of Pathology in collaboration with the department of Physiology, Rajiv Gandhi Medical College, Kalwa. Before proceeding for the study, the plan of the study was submitted to the local 'Ethical Committee' & was approved. The study was explained to the subject & consent was taken.

**Study Area:** This study was restricted to the students studying in the same college.

**Study Design:** Cross sectional study

**Study Population:** It comprised V semester medical students studying in the same medical college.

**Data Source:** Roll call register

**Tools of Data:** Planned questionnaire, pre- & post-test marks

**Sample size:** 48

**Statistical Tests:** 't' test (paired, unpaired), Z test

### **Procedure:**

The study was conducted in the Department of Pathology, Rajiv Gandhi Medical College, Kalwa. We went through the Maharashtra University syllabus of Pathology & Physiology and identified the areas of overlap between the two subjects. Out of these areas, Jaundice with

Liver function test & Diabetes Mellitus were chosen. The project was explained to the students & they were motivated to participate in it. The students were divided into two equal groups, Group A & Group B. For first topic (Jaundice & Liver function test), group A was given traditional & group B was given integrated lecture. Reverse was done for second topic (Diabetes Mellitus). Both the traditional lectures were taken with OHP whereas the integrated lectures were taken with LCD presentation. In both the cases, pre-test & post-test were given with prevalidated MCQ's. Also, the student's feedback was taken by giving them prevalidated questionnaire containing both close & open-ended questions. Students were told not to reveal their identity to make this exercise honest & free from bias.

## Result & Analysis

For the comparison between pre-test & post-test of both lecture topics, the 't' test (paired) was used. The statistical value for traditional was found to be 6.704 & for integrated lecture was found to be 8.100. Both the values were highly significant.

For the comparison between post-test of integrated & traditional lecture 't' test (unpaired) was used. The statistical values for post-test 1 & post-test 2 were found to be 0.756 & 0.332 respectively. These values were non-significant.

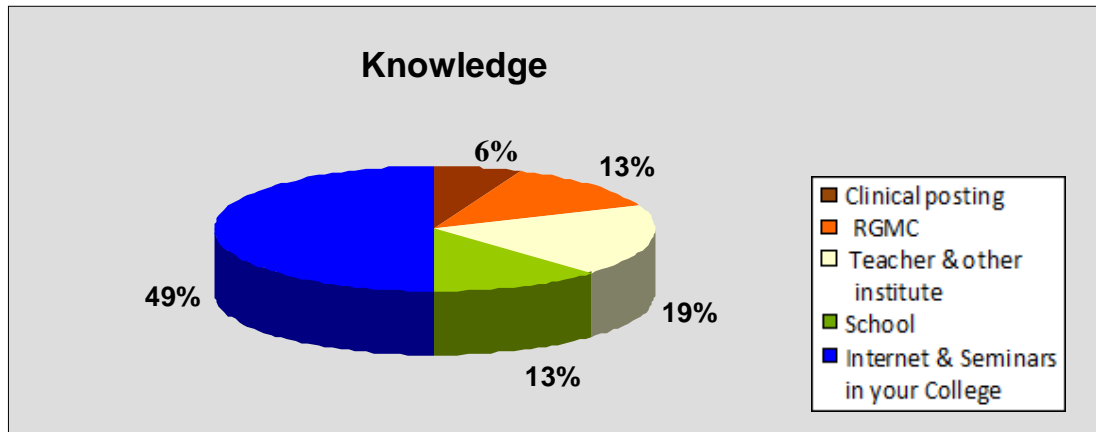
The following table shows the mean, mean  $\pm$  1.96 standard deviation and mean  $\pm$  1.96 standard error for both the pre-test & post-test of traditional & integrated lectures:

Factors	Mean	Mean $\pm$ 1.96SD	Mean $\pm$ 1.96SE
PRETEST 1(TL)	12.05	6.49 - 17.61	10.83 - 13.26
PRETEST 1(IL)	13	6.24 - 19.76	11.52 - 14.48
POSTTEST 1(TL)	18	14.25 - 21.75	17.14 - 18.86
POSTTEST1(IL)	18.71	11.78 - 25.63	17.03 - 20.38
PRETEST2 (TL)	19.11	13.36 - 24.86	17.19 - 21.03
PRETEST2 (IL)	18.2	12.67 - 23.73	16.45 - 19.95
POSTTEST2 (TL)	24.67	21.89 - 27.49	23.74 - 25.59
POSTEST 2 (IL)	24.25	17.48 - 31.02	21.86 - 26.64

Questions in the student's feedback questionnaire were analyzed statistically by using 't' test (paired, unpaired) & 'Z' test.

### 1. Previous knowledge of integrated teaching methodology –

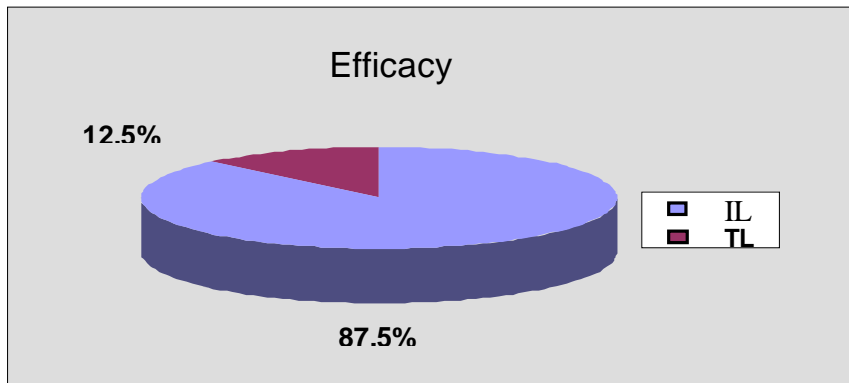
40% students were unaware of integrated teaching methods whereas 60% students had heard about integrated teaching.



The above pie diagram shows the % of different sources. 49% students heard about it from Internet, seminars in college & teachers from other institute were significant. Other sources like clinical posting, school, in same institute were insignificant.

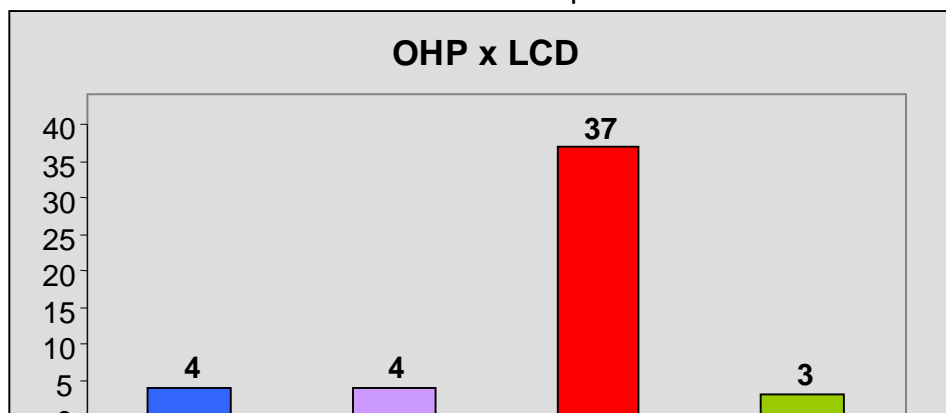
### 2. Efficacy of integrated lecture –

87.50% students thought that integrated lecture is more effective than traditional & 12.50% thought traditional is better than integrated.



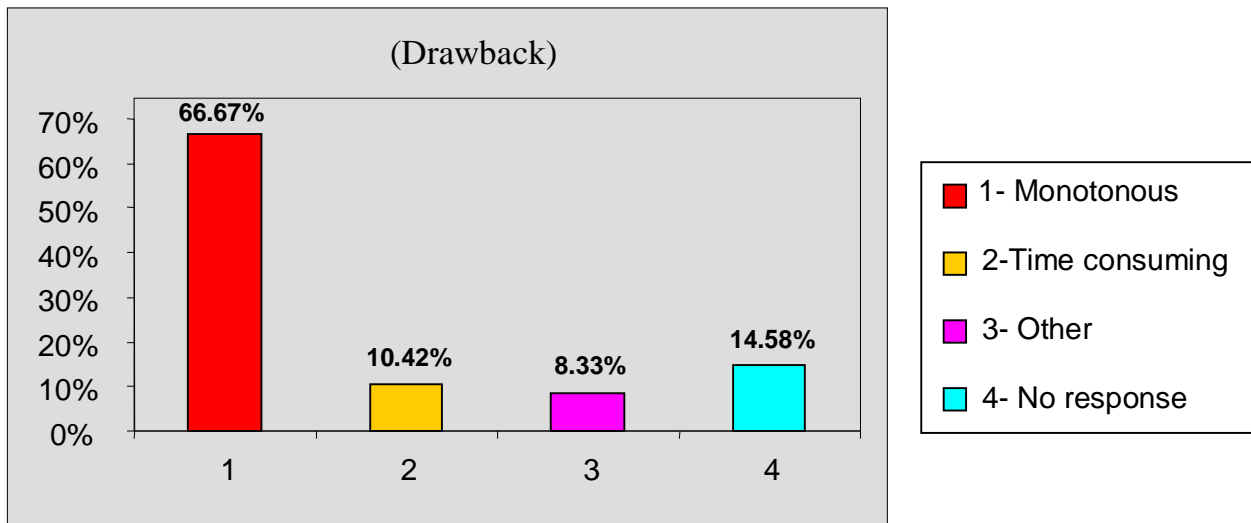
### 3. Efficacy of LCD presentation –

The following table shows the frequency of effectiveness of OHP & LCD. LCD was chosen by maximum students i.e. 37 students compare to OHP.



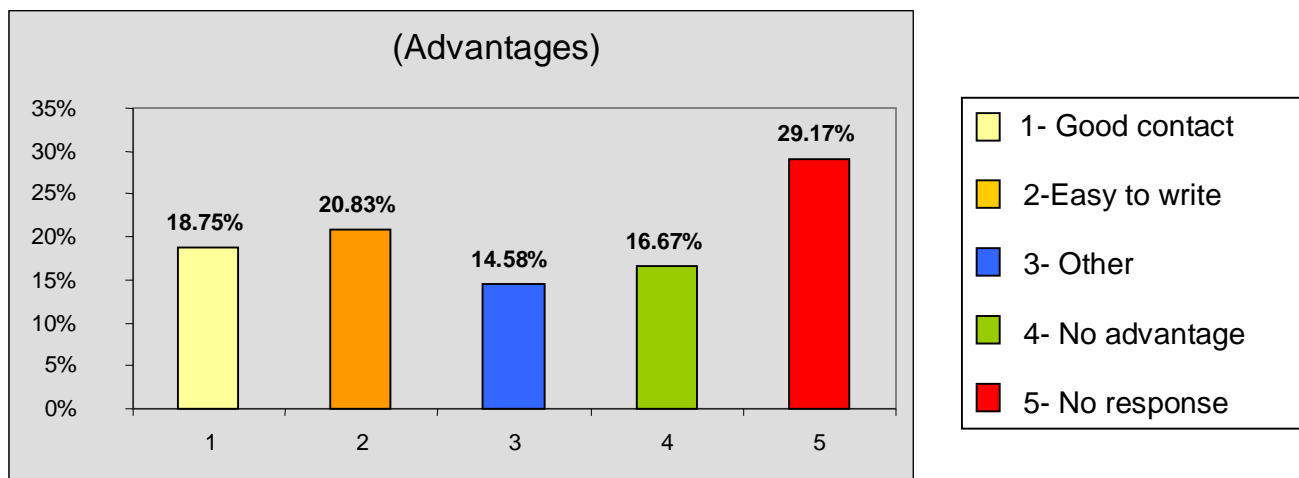


#### 4. Drawbacks of traditional lecture –



The above bar diagram shows percentages of various drawbacks. Maximum percentage i.e. 66.67% students' felt that traditional lectures are monotonous, boring & acts as sedative. 14.58% students did not respond to question. 10.42% students felt that they are time consuming & problems with illegible handwriting. 8.33% gave other drawbacks like mechanisms especially are difficult to understand, knowledge of faculty etc.

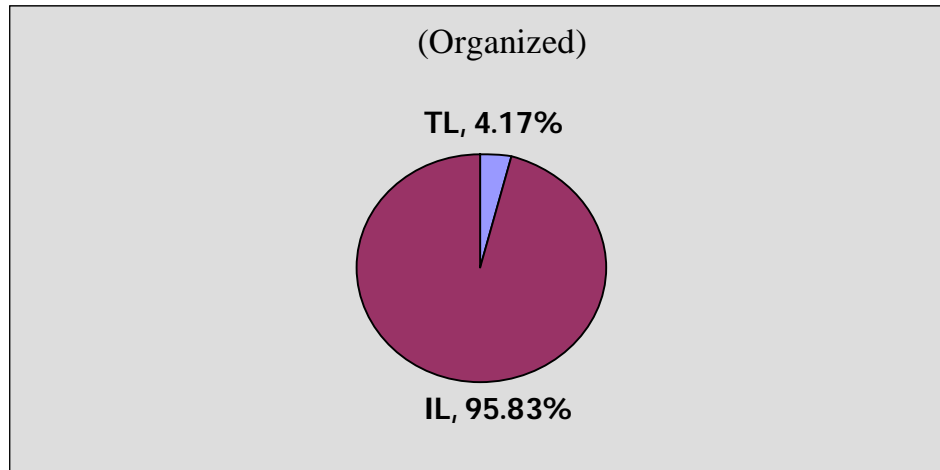
#### 5. Advantages of traditional –



The above bar diagram shows percentage of various advantages. Maximum i.e. 29.17% students did not respond this question. 20.83% students' felt that it is easy to write notes & is less time consuming. 18.75% students felt that there is good personalized contact & understanding. 16.67% students felt that there is no advantage of traditional lecture over integrated lecture. 14.58% students gave other advantages like more details covered & less effort for teachers.

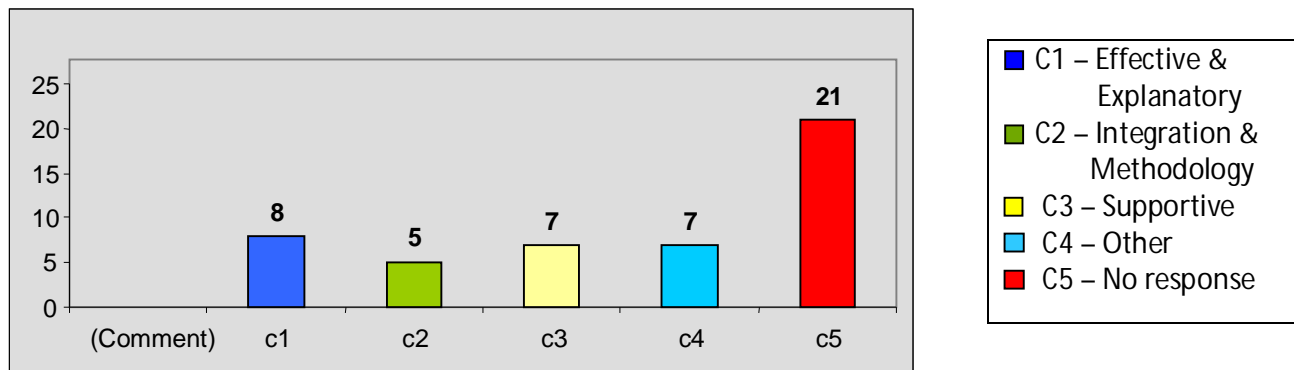
## 6. Periodic Organization of integrated lectures –

95.83% students wanted more integrated lecture & felt that it gives clearer picture of disease, progress & its management whereas 4.17% students didn't want it.



The above pie diagram shows the ratio of percentage of students who would like to have the integrated & traditional lectures.

## 7. Comments about integrated teaching methodology-



The above bar diagram shows the frequency of various comments given by the students over the integrated lecture. Most of the students i.e. 21 did not respond to the question. 8 students said that it is more effective & more explanatory. 5 students felt that it is more needed in third year & more supportive method. 7 students would like to have more animation & pictorial presentation. 7 students felt it is time consuming & requires time to write notes & topics should be interesting.

## Discussion

This study was conducted to demonstrate the utility & feasibility of an integrated learning process. Students found the integrated learning programme to be a useful, feasible method of learning. However, the statistical comparison between the evaluation after integrated & traditional lectures had not shown significant impact of integrated lectures. But certainly students felt the need for integration.

**"Some people talk in their sleep. Lecturers talk while other people sleep."**

This was the effect of traditional lecture for most of the students. They feel that traditional lectures are more boring, monotonous & good sedatives whereas integrated lectures are more explanatory, less boring, interesting & supportive. Most of the students would like similar integrated lectures to be organized periodically, especially for certain topics in third year.

This study was an attempt to improve the quality of medical education with the innovative curricular strategy. The lacuna of the study was the small sample size. Similar study with larger sample size will be more productive. Also, we could not cover the long-term impact of integrated teaching methodology. But the integrated teaching would surely reduce the fragmentation of medical course; prevent repetition, waste of time & rationalization of teaching resources. It will also promote the interdepartmental collaboration & students will learn to apply their knowledge to clinical practice.

### Reference:

1. Joglekar S, Bhuiyan P S, Kishore S. Integrated teaching – Our Experience  
Journal of Post-graduate Medicine 1994; 40: 231-232. <http://www.jpjgmonline.com>
2. Joglekar S S, Bhuiyan P S, Pandit D D. Integrated Teaching in The Art of Teaching  
Medical Students Bhuiyan P S et al. 2<sup>nd</sup> Edition, Mumbai, Medical Education  
Technology Cell, Seth GSMC & KEM Hospital, 2002, Pg 305-312.
3. Sharma R C. Methods of Enhanced Learning: An Interactive T-L Approach Indian  
Journal of Physiology & Pharmacology Supplement 2006; 52 No. 5 27

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### ANNEXURE:

Student feedback form

1. Have you heard about the integrated teaching methodology before attending the session?  
Yes/No
2. If Yes, Where did you learn about it?
  - a. In clinical posting
  - b. In RGMC
  - c. From teachers/other institute
  - d. In school
  - e. On internet
  - f. Seminars in your college
3. Is integrated teaching more effective than traditional lecture?  
Yes / No
4. Is OHP presentation more effective than LCD presentation or vice versa?

5. What are the drawbacks of traditional lectures with OHP presentation?
6. What are the advantages of traditional lectures with OHP presentation?
7. Would you like similar integrated lectures to be organized periodically?
8. Is integrated teaching helpful in projecting a more complete picture of understanding the disease process & its management?  
Yes / No
9. Any comments/ suggestions about integrated teaching methodology.

### **(3) EVALUATION OF HYPONATREMIA IN HEART FAILURE PATIENTS ADMITTED IN CRITICAL CARE UNIT: SINGLE CENTRE EXPERIENCE.**

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1. Professor
2. Associate Professor
3. Assistant Professor
4. Resident

U N Mehta Institute of Cardiology & Research Centre,  
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**Address for Correspondence : Dr. J. R. Rawal,**  
Professor & Head,  
U N Mehta Institute of Cardiology & Research Centre,  
Civil Hospital, Ahmedabad.

#### **Abstract :**

##### **Background & Objectives :**

Hyponatremia is a very common electrolyte disturbance in hospitalized patients of heart failure. Clinical profile of such patients with hyponatremia and common co-morbidities are subjects of current study where we made an attempt to evaluate severity, symptoms and in hospital outcome of such patients.

**Methods :**

All patients presented to intensive coronary care unit (ICCU) with decompensated congestive cardiac failure (CCF) during three months period in 2012 were included in the study.

**Result :**

Out of total 1184 ICCU admissions, 650 were presented with decompensated heart failure. Serum sodium levels less than 135 mmol/L was found in 334 patients while severe hyponatremia less than 110 mmol/L was seen in 5.3%. Symptomatology ranged from mild irritability and intractable vomiting to seizures and unresponsiveness on neurologic evaluation. Male preponderance (64.40%) was noted. Common co-morbidities were hypertension (64.22%), Diabetes mellitus (32.41%), Ischemic Heart Disease (67.42%) and valvular heart disease (10.5%). Two diuretic molecules were co-administered in (39.70%). Females tolerated different grades of hyponatremia better than males with fewer symptoms and less mortality.

**Conclusion :**

Hyponatremia is more common in male patients presenting with CCF however, female patients with CCF have less mortality and fewer symptoms. There is a direct relation of serum sodium concentration and in hospital mortality.

**Key words :**

Hyponatremia, congestive cardiac failure, diuretics.

**Introduction :**

Hyponatremia is found to be fairly common biochemical abnormality in CCF patients. Primary mechanism is dilutional hyponatremia where arginine vasopressin (AVP) is secreted without any triggering signals of osmolality change. Severe heart failure causes hormonal release of AVP via baroreceptor mediated signals. Studies have shown that prevalence of hyponatremia (defined as less than 135mmol/L) ranges from 15 to 30% in different chronic care hospital settings. The incidence is much common in elderly and female patients with some impairment of renal or cardiovascular functions. The symptoms of hyponatremia develop according to severity and the speed of fall in sodium concentration. Reducing plasma concentration of sodium generates an osmotically driven water movement into brain cells resulting into brain oedema, raised intracranial pressure and neurologic symptoms. When hyponatremia builds up slowly brain cells have regulatory mechanism operating in few hours to days which prevents brain oedema and hence fewer symptoms. Organic solutes like glutamate, taurine, myo-inositol and glutamine are involved in counter regulatory transport across the cell membrane, thus ameliorating cellular oedema in brain during chronic stages of hyponatremia. Hyponatremia predicts higher mortality in congestive cardiac failure; community acquired pneumonia and hospitalized patients in general. Hyponatremia has wide variety of causes, commonest being overzealous diuretic usage combined with salt restriction in hypertensive individuals & CCF patients. While acute hyponatremia symptoms may progress to death, the treatment for hyponatremia is also carrying serious risk of Central Pontine Myelinolysis (CPM). This complication is result of rapid correction of hyponatremia. Brain cells require time to adapt to

changes in osmotic environment. Pons have very tight grid arrangement of glial tissue and axons hence, Pons is the seat of major injury of osmotic imbalance.

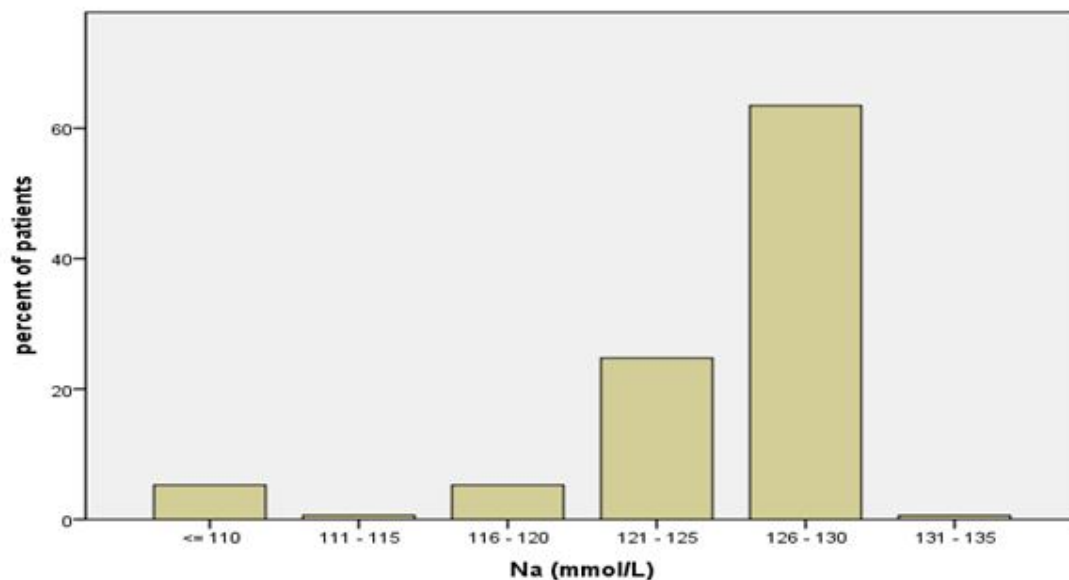
Data regarding incidence of hyponatremia in heart failure patients and ICCU patients are very scanty in our country. The present study was aimed at getting clinical features, aetiology and in hospital outcome of variable digress of hyponatremia in CCF patients admitted in ICCU.

### Methods :

1184 Consecutive patients admitted in our hospital with symptoms of decompensated congestive cardiac failure between 1<sup>st</sup> June, 2012 to 30<sup>th</sup> Sept., 2012 were evaluated. Relevant history and clinical features were recorded including past medical history, drug history, examination findings and volumic status was recorded. Skin turgor, pulse rate, postural blood pressure, oedema legs, basal craps in lung fields, jugular venous pressure etc. were subjectively evaluated to record volumic status of patients and accordingly, hypervolaemic, euvolaemic and hypovolaemic status categories were designated to classify patients of CCF. Routine blood and urine tests including complete blood counts, renal function tests, electrolytes, liver function tests, chest radiographs, transthoracic echocardiography, troponin I levels, CT scan (if clinically indicated) were performed in all these patients. All patients were treated for hyponatremia as per hospital protocol.

Investigations were repeated during hospital stay as per clinical judgment of caretaker physician. Serum sodium concentration was measured on 'Cobas Integra 400' fully automated 'Roche Diagnostics' machine.

Normal laboratory range is 136 to 145 mmol/L.



### Results :

Total 1184 patients were admitted to ICCU of our hospital between 1<sup>st</sup> June, 2012 upto 30<sup>th</sup> September, 2012. Out of these, 650 patients presented with clinical features of congestive cardiac failure. Total of 334 patients have serum sodium concentrations less than 135mmol/L.

The mean age of patients having hyponatremia and CCF was 44.25 years. Male preponderance was noted 64.40%. The mean sodium level on admission was 122.95 mmol/L and pre-discharge

repeat levels were done with mean value of 131 mmol/L. Hypervolemic status was seen in 71.42% and 26.20% patients were found euvolemic.

Mild hyponatremia was asymptomatic. However, in CCF patients even levels below 125 mmol/L were asymptotically tolerated. Nausea and malaise were frequent below 120 mmol/L and headache, lethargy, confusion, disorientation prevail after levels reach below 115 mmol/L. Most common presentation was drowsiness at 110 mmol/L and seizures, coma was found at 105 mmol/L levels.

Drowsiness, irrelevant talk and poor response to verbal stimuli without any focal neurologic deficit were the (68.71%) most common central nervous system (CNS) presentations. Three cases had significant loss of fluid with intractable vomiting. All patients with neurological symptoms were evaluated for structural damage on CT Scan. No structural neurologic abnormalities were detected in CT Scan.

The common co-morbid conditions were hypertension (64.22%) and Diabetes mellitus (32.41%) and ischemia heart disease (67.42%), renal failure (13.60%). Co-existing hypokalemia was noted in 7.10%. Two different molecules for diuretics were used simultaneously in 39.70%. Metolazone use was seen in 3.7%. 28 patient succumbed to their heart failure. Out of them, 16 had sodium levels below 105 mmol/L. Female patients had lower mortality of 18.48% in severe hyponatremia patients ( $< 105$  mmol/L  $\text{Na}^+$ ) while mortality in males with severe hyponatremia was 57.14%.

## **Discussion :**

Heart failure patients frequently present with hyponatremia as a part of water retention and oedema. This study shows higher prevalence of this biochemical abnormality (51.38%) in patients presenting with CCF. However, these patients tolerate it better upto 120 mmol/L with minimal symptoms attributable to hyponatremia. The prevalence is more in males as compared to females. This reflects the sex distribution of CCF patients presenting in critical care unit. Chronic hyponatremia is common in postmenopausal women. However, in those patients having CCF as presenting syndrome male dominance is seen. Sodium concentration less than 135 mmol/L was associated with increased 30 day mortality or recurrence of myocardial infarction in non ST segment elevation myocardial infarction (NSTEMI). Similarly, risk of death and heart failure are higher among ST segment elevation myocardial infarction (STEMI) patients who had hyponatremia on admission. Activation of arginine vasopressin (AVP) may impair water excretion and result in dilutional hyponatremia in STEMI patients with acute left ventricle (LV) dysfunction.

Mortality rates have been reported in wide range from 33% to 86% in elderly patients and 20% in an Indian report by Rao MY. However, our study reports 8.38% mortality rates in CCF patients with hyponatremia. Previous mortality data in hyponatremia has included syndrome of inappropriate antidiuretic hormone secretion (SIADH), cirrhosis and renal causes also. In fact, MY Rao et al reported SIADH and old age ( $> 72$  yrs) as major demographic factors in their group and hence higher mortality. The present study focused only heart failure patients with hyponatremia where mortality is lower as compared to other studies possibly because mean age was less (younger population) and slow development of chronic hyponatremia in CCF patients is better tolerated with fewer symptoms and less mortality as compared to other

group containing older patients and SIADH patients. Severe hyponatremia less than 105 mmol/L has very high mortality rates (57.14%) and seizures were seen in 8 cases (50% of those who succumb to hyponatremia).

Drug induced hyponatremia needs special mention here. Many studies have documented thiazide diuretics as a major cause. In present study two different molecules of diuretics were used in 30.70% cases. All of them received furosemide by intravenous route. Co-existing hypokalemia (7.10%) also complicates the course of the disease. Though joint national committee (JNC) VII recommended diuretics as first line of drugs for treatment of hypertension, a word of caution is expressed here because of frequent occurrence of hypernatremia following its indiscriminate usage. CCF patients continue to receive diuretics however, dose modification or withdrawal of such drugs may be done when systemic congestion comes down to bare minimum levels and angiotensin converting enzyme (ACE) inhibitors reach up titration to their optimal recommended dosages.

This study does not look into the methods of correction of hyponatremia and the responses to treatment. It highlights very common association of dilutional hyponatremia in CCF and emphasizes more usage of vasodilators like ACE inhibitors and minimum use of diuretics because diuretics may add to the burden of hyponatremia in CCF. In Congestive Cardiac Failure (CCF), higher mortality is expected due to ongoing disease severity or possibly due to mismanagement.

Further studies are needed with controlled prospective design so as to include all severity grades of hyponatremia patients to find out mortality rates linked with severity of hyponatremia, specific subsets of cirrhosis, CCF etc.

## Reference:

1. Adroge, HJ, Madias NE. Hyponatremia. *N Engl J Med* 2000;342:1585-9
2. Klein L, O'Connor CM, Leimberger JD, Gattis – Stough W, Pina II, Felker GM, et al. Lower serum sodium is associated with increased short-term mortality in hospitalized patients with worsening heart failure: results from the outcomes of a prospective trial of intravenous milrinone for exacerbation of chronic heart failure (OPTIME\_CHF) study. *Circulation* 2005;111:24554-60
3. Chung H-M, Kluge R, Schrier RW, Anderson RJ, Clinical assessment of extracellular fluid volume in hyponatraemia, *Am J Med* 1987;83:905-8.
4. Goldberg A, Hammerman H, Petcherski S, Nassar M, Zdrovovak A, Yalonesky S, et al. Hyponatremia and long-term mortality in survivors of acute ST-elevation myocardial infarction. *Arch Intern Med* 2006; 166:781-786.
5. Singla I, Zahid M, Good CB, Macioce A, Sonel AF, Effect of hyponatremia (<135 mEq/L) on outcome in patients with non-ST-elevation acute coronary syndrome. *AM J Cardiol* 2007: 100:406-408.
6. Nzerue CM, Baffoe-Bonnie H, Falana B, Dai S. Predictors for outcome in hospitalized patients with severe hyponatraemia. *J Nat Med Assoc* 2003; 95:335-43.



7. MY Rao, U Sudhir, T Anil Kumar, S Saravanan, E Mahesh, K Punith. Hospital-Based Descriptive Study of Symptomatic, Hyponatremia in Elderly Patients. JAPI 2010; 58:667-669.
8. A Goel, OP Kalra. Hyponatremia in Older Individuals. JAPI 2010; 58:663.
9. Tada Y, Nakamura T, Funayama H, Sugawara Y et al. Early development of hyponatremia implicates short and long term outcomes in ST elevation Acute Myocardial Infarction. Circ J 2011; 75 : 1927 – 1933.

#### **(4) POST MORTEM STUDY OF HEART IN CASES OF SUDDEN CARDIAC DEATH USING TRIPHENYL TETRAZOLIUM CHLORIDE AND HAEMATOXYLIN & EOSIN STAIN**

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#### **ABSTRACT:-**

##### **Background:**

Myocardial infarction (MI) is the most common cause of death for which the largest number of autopsies are done.

##### **Aims:**

A clinical study was done to determine those cases numerically smaller but medico-legally more important in which some unnatural event like a road accident, is suspected to die due to acute myocardial infarction. The likelihood of identifying an early infarct minimizes false positivity in cases of sudden death.

##### **Materials and Methods:**

A histochemical study on 100 hearts was carried out with using haematoxylin & eosin as a routine stain and Triphenyl tetrazolium chloride as gross marker, the latter can detect infarcts

of less than 6 hours of age which help to assess the actual age of the infarct and confirmation of myocardial infarction as the cause of demise.

### **Results:**

68% of cases showed positive result with TTC staining while only 25 % of cases showed positive result with H & E stain. 75% of cases showed the age of infarction within 5 to 8 hours which made possible to detect early infarction by TTC staining. In 82.3% of cases gross examination did not reveal any evidence of infarction, but were revealed by TTC staining.

### **Conclusion:**

Triphenyl tetrazolium chloride is the detector of earliest changes of infarction in comparison to gross and microscopic changes in the infarcted heart.

### **Keywords:**

Triphenyl tetrazolium chloride, early infarct.

### **Introduction:-**

Ischemic heart disease (IHD) is the leading cause of death worldwide for both men and women. In more than 90% the cause of myocardial infarction is reduced blood flow due to obstructive atherosclerotic lesion in the coronaries, thus IHD is also termed as coronary artery disease (CAD) or coronary heart disease (CHD).<sup>1</sup> CAD is by far the most frequent cause of sudden and unexpected deaths which constitute a significant portion of autopsies conducted by forensic pathologists in our country, because these deaths appear in apparently healthy without any diagnosed disease that can be attributed to the cause of death, or the period of illness before the supervening of death is so short that the disease cannot be diagnosed early.

The realization of the need for establishing the diagnosis of myocardial infarction in the initial 8 hours, where definite evidence of infarction is lacking has propelled a number of studies on the histochemical, electron microscopic and fluorescent microscopic changes. Also many times significant stenosis of coronary arteries is encountered in deaths due to some other natural disease, in these cases the query always arises whether myocardial ischemia might have contributed to death.<sup>2</sup> With these considerations it was thought worthwhile to study myocardial ischemic changes, with gross histochemical staining and routine staining methods. The histochemical method for studying the enzyme reaction in the myocardium, to differentiate between the healthy myocardium and the infarcted myocardium is by far the most sensitive method for diagnosing early myocardial ischemia and is comparatively simple as compared to other methods. The present study has been carried out with utilization of Triphenyl tetrazolium chloride as gross marker and haemotoxylin and eosin as a routine staining method on dead myocardium to separate it from viable myocardium to assess the actual age of myocardial infarction and to confirm myocardial infarction as the only cause of death.

### **Materials and Methods:-**

The present study was carried at the Department of pathology and department forensic medicine and toxicology during the period of August 2009 to August 2011 in M. P. Shah medical

college, G. G. hospital, Jamnagar. Total 100 cases were studied. The study was approved by institutional ethical committee, prior to inclusion of cases in the study. Detailed history and post-mortem (PM) findings of heart of all cases were collected. PM study was carried out in clinically suspected/ confirmed cases of MI. In addition 3 cases of sudden death due to other causes were also included in the study as negative control. Cases showing signs of decomposition were not included.

Hearts obtained from the test cases were subjected to meticulous gross examination of all three coronary vessels and their major branches by serial transverse sectioning of vessel to note the degree of calcification, stenosis, and presence of thrombus or hemorrhage into an athermatous plaque. This was done by cutting transverse sections of the coronaries and their main branches at 3 mm intervals. Complete transverse slice of ventricular myocardium from sites containing evident or suspected fresh infarct by gross appearance; significant narrowing of vessel supplying the area etc were subjected to TTC macro test. In absence of any of these pointers, multiple slices were studied. Routine microscopic examination by H & E stain of suspected infarct or peripheral area of visible fresh infarct or fresh and old infarction delineated after TTC test was also undertaken.

### **TTC Macro Test:**

Transverse Slicing of Myocardium<sup>3, 4, 5, 6, 7</sup>: 1.5 cm thick transverse slices were cut from ventricular part of fresh heart (without formalin fixation) from apex of heart (Figure 1). This method permits best identification of infarction as well as easy location and documentation of grossly discernible myocardial infarcts. It is indicated whenever ischemic heart disease is in question and quantification of infarct size is desirable. Select the slices which is likely to be a contain infarcted area as per guidelines mentioned above were dipped in running water before incubating to remove excess of blood from the surface of the slices but were not allowed to soak or macerate in water as the enzyme reaction is purely a surface phenomenon and prolonged washing elutes the contents of the superficial cell thus giving a false reading. The slices were incubated in 1% solution of 2, 3, 5 Triphenyl Tetrazolium chloride (TTC) for 20-30 minutes, at room temperature or some authors advocate incubation at 37-40<sup>0</sup> C. The incubating fluid was prepared by dissolving 1 gm of TTC powder in 100 ml of phosphate buffer at pH 8.5. The staining was carried out in appropriate sized (preferably glass cylindrical of 7.5 diameter and 9.5 cm height) wide mouthed container with screw cap, which can hold a complete transverse slice. During the process of incubation the container is to be closed by its screw cap and kept in a dark place. As exposure to air and light will make the solution lose its potency. TTC solution was freshly prepared every time and was used within half an hour. The slice was to be immersed completely in the solution during the process. The upper surface of it being at least 2 cm below the fluid level. We found that 100ml of 1% solution poured into the above sized container fulfils this criterion, when one slice of heart was immersed into it. The most important precaution of all was to regulate the pH of incubating fluid at 8.5 otherwise the formation of formazan pigments will be unsatisfactory. For better results the slice should be turned over once or twice during the process to discourage stagnation of the reagent and causing excessive deposition of the dye on the surface of the heart slices thereby producing artifacts. The slices were turned over after 15 minutes to prevent prolonged contact of the tissue with the bottom of jar. At the end of the

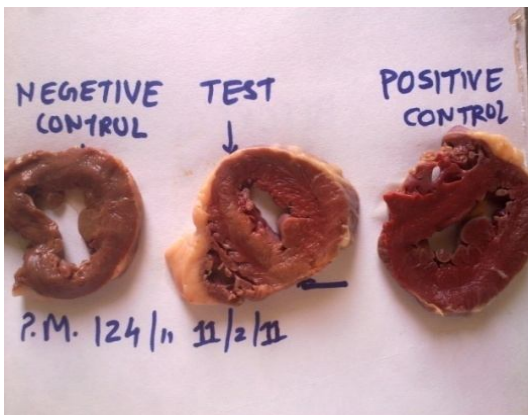
staining procedure the slice was transferred to a jar containing 10% formal saline. This will halt the reaction immediately in addition to fixing the tissue.

**Interpretation:**

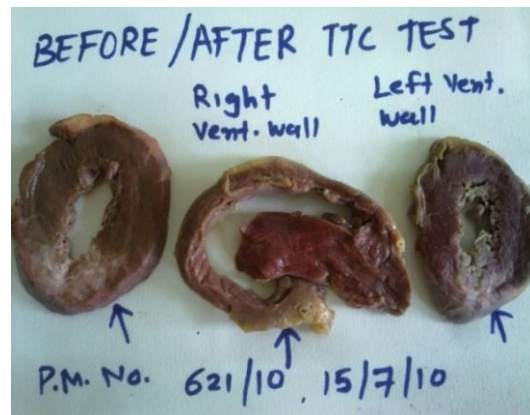
On completion of the staining the infarcted myocardium will show up as pale pink colored area (Figure-2, 3) as against the bright red colored normal myocardium (Figure-4). Even old infarcts and scars get delineated (Figure-5). In absence of infarction, both the surface of the slice will show uniform bright red coloration.



**Fig 1** Method of dissection of TTC staining transverse slicing of Heart 00



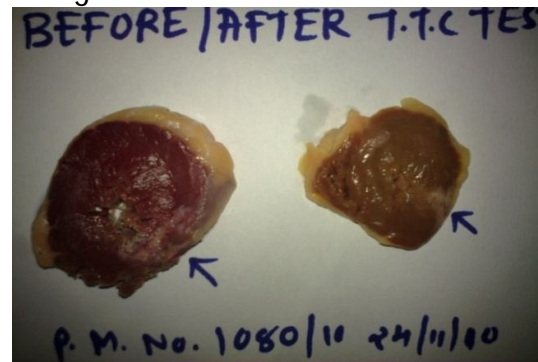
**Fig 2** TTC stained heart slice - Fresh infarct seen as pale stained area



**Fig 3** TTC stained heart slice - Old infarct in left ventricle & Fresh infarct in right ventricle



**Fig 4** TTC stain - Normal myocardium uniformly stained



**Fig 5** TTC stained slice - Old infarct seen as unstained area

**Results:****Table No. 1 Result of TTC stain and H&E stain of myocardium slice**

Age Range	Total	Positive TTC stain	Positive H&E stain	Negative TTC stain	Negative H&E Stain	Doubtful TTC stain	Doubtful H&E Stain
0-10	0	0 (0%)	0 (0%)	0	0	0	0
11-20	3	1 (33.3%)	0 (0%)	1	2	1	1
21-30	3	2 (66.7%)	1 (33.33%)	1	2	0	0
31-40	21	13 (61.9%)	0 (0%)	6	19	2	2
41-50	21	15 (71.4%)	3 (14.28%)	5	17	1	1
51-60	37	25 (67.6%)	15 (40.54%)	11	21	1	1
61-70	10	8 (80%)	4 (40%)	2	6	0	0
71-80	5	4 (80%)	2 (40%)	1	3	0	0
81-90	0	0 (0%)	0 (0%)	0	0	0	0
91-100	0	0 (0%)	0 (0%)	0	0	0	0
Total	100	68 (68%)	25 (25%)	27	70	5	5

Table No. 1 shows early ischemic changes in myocardium were detected by positive TTC staining in 68% of cases. Amongst these 80% results were observed in 7<sup>th</sup> and 8<sup>th</sup> decade. Only a case (33.3%) of young adult was shown positive result with TTC staining. In present study only 25 cases out of 100 were detected by H & E stain. 40% of result was obtained in age group 51-80 years indicates low sensitivity of H & E stain in selected sample type and post myocardial infarction survival duration is longer in middle to older age group.

**Table No. 2 Correlation of survival time with TTC staining**

Period of survival (Age of infarction)	Cases
0-4 hours	04 (5.9%)
5-8 hours	51 (75%)
8-12 hours	06(8.8%)
Not known	07(10.3%)
Total	68

Table No. 2 shows maximum (75%) number of cases showed the age of infarction within 5 to 8 hours which made possible to detect early infarction by TTC staining in comparison to gross and microscopic changes in the heart following myocardial infarction. The period of survival was not known in 7 cases because reliable history was not available.

**Table No. 3 Signs of fresh infarct on gross examination**

Gross finding	No. of case
Grossly infarct present	12 (17.6%)
Grossly infarct absent	56 (82.3%)
Total	68

Table No. 3 shows maximum (82.3%) cases of which gross examination did not reveal any evidence of infarction, which were revealed by TTC staining. So, TTC is found to be useful test to detect the early infarct as well as to delineate the existed infarct. The periods of survival assessed from the onset of the episode of chest pain to the death, ranged from 1 hour to 12 hours. The attempt was made to correlate the result of TTC with supposed period of survival.

### **Discussion:-**

The absence of gross as well as microscopic changes before appearance of neutrophil at the scene of infarct, which is estimated to take minimum of 6-8 hours in term of post-infarction survival time, has been a major hurdle in establishing the cause of death in cases of early death due to myocardial infarction. As Nitro-Blue and TTC have almost same interference and present study was experimental study, studies regarding Nitro-Blue have also been included because less work has been done on TTC application.

### **TTC Results:**

Present study reveals fresh infarct in 82.37% autopsy cases using TTC stain, which is closely correlates with Agdal<sup>8</sup> study where he found fresh infarct in 75% of cases, indicate more number of fresh infarct can be detected by using TTC.

### **Grossly Visible Infarct:**

In present study there were 68 TTC positive test, amongst them 56 specimen of heart did not show infarction on macroscopic examination. After application of TTC, infarcted areas were well visualized. In 12 hearts in which macroscopically infarcted area were seen, after application of TTC these areas were better delineated. Finding of the present study is in accordance with Neural et al<sup>9,10</sup>, Nachals and Shnikta<sup>5</sup>, Ramkissoon<sup>6</sup>, Brody et al<sup>4</sup> and Mc Vie<sup>11</sup>

### **Period of Survival:**

Another issue to be considered was the detection of myocardial ischemia in relation to the period of survival and also whether these techniques which outline experimental myocardial infarcts, before the gross or microscopic changes manifest, can be used to assist pathologists in the recognition of early or small infarcts in the human subject.

The earliest infarcts that was detected by TTC methods adopted in present study is of 1 hour duration (old), These observations were closest to those made by Fine<sup>12</sup>, where he found loss of enzyme activity from 1½ hours after the onset of symptoms using the Nitro B.T. method. Also, It coincided with the findings of Andersen and Hensen<sup>13,14</sup> who detected localized subendocardial infarctions of 1 hour duration. They however reason that subendocardial infarctions presumably had a symptoms free period. It was also the hypothesis of Gregersen<sup>15</sup> that all infarcts start in the subendocardial zone.

We might deduce that perhaps the subject had survived for a longer period of time a transmural infarction might have been demonstrated due to the loss of enzyme activity. This discrepancy in the time required for demonstration of loss of enzyme activity could also be due to unreliability of fixing the actual event of infarction to coincide with onset of pain or any other clinical symptoms.

The age of an infarct is notoriously difficult to establish in the human, as the onset of clinical symptoms, however dramatically abrupt, are often much later than the onset of the pathological lesion precipitated by a coronary occlusion. In animal experiments, a coronary vessel can be ligated at zero time and serial sacrifices made at different intervals to gain an accurate estimate of the age of the infarct. In the human, the time of chest pain and shock cannot be used in a similar fashion. When a victim of coronary disease dies say, eight hours after the onset of acute symptoms, though one might expect an early infarct to be visible on histological or histochemical examination, not frequently a well-demarcated yellow or tigroid area of necrosis is present; which must be several days old.

### **Conclusion:**

This study shows that the Maximum number of early myocardial infarction (less than 6 hour of duration) can be detected by TTC method. The technique of this procedure is quite simple and easy to perform. The reagents required are not out of the reach (economically) for medium sized laboratories. As regards interpretation of results, the TTC macro-method did not pose any problems. While carrying out this type of study to minimize false results, selection of proper sample type is very important. An attempt should be made to establish TTC macro method at all the hospitals including primary centers where the postmortems are conducted.

### **Acknowledgement:**

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**Conflict of Interest:** - None Declared.

### **References:-**

1. Ramzi S. Cotran, Vinay kumar, Abbas, Fausto, and Aster Robbin's: Pathological basis of disease; USA: Elsevier, PA. 2010, 8<sup>th</sup> edition. p.529-87.
2. Strong JP, McGill HC: The Natural History of Coronary Atherosclerosis. Am J Pathol.1962; 40: 37-49.
3. Kothabagi RB, Apte VV, Pathak PR: POSTMORTEM DIAGNOSIS OF EARLY MYOCARDIAL INFARCTION. MJAFI. 2000; 56(2): 99-102.
4. Brody GL, Belding AW, Elding MR, Feidman SA: The identification and delineation of myocardial infarcts. Arch Pathol. 1987; 84:312- 17.
5. Nachlas MM, Shinka TK: Macroscopic identification of early myocardial infarction by alteration in dehydrogenase activity. Am Pathol. 1963; 42:379-405.
6. Ramkissoon RA: Macroscopic identification of early myocardial infarction by alteration in dehydrogenase activity. J Clin Pathol.1966; 19(5):479-81.
7. Ludwig J: Current method of autopsy practice: HEART AND VASCULAR SYSTEM: Philadelphia, Saunders. 1979, 2<sup>nd</sup> Edn. p.51-92

8. Niels Agdal, Mogens Thyge Anderson The value of succinate dehydrogenase stain in the postmortem diagnosis of early acute myocardial infarction: A forensic study.
9. Neoral L, Kolin A, Kodousek R, Kvasnicka J: A study about early stages of experimental ischemia and myocardial infarction in dogs. Acta Univ. Palak Olomucensis. 1956; 11:127-33.
10. Neoral L, Kolin A, Kodousek R, Kvasnicka J, Beitragzur Kenntnis der Fruhstadien der experimental Myokardischamie. Zentralbl. Allg. Path.1959; 99:239-44.
11. Mcvie JG: Postmortem detection of inapparent myocardial infarction. J Clin Pathol. 1970; 23:203-09.
12. Fine G, Moreales A and Screpella JR: Experimental myocardial infarction. A histochemical study. Arch Path. 1966; 82: 4-8.
13. Andersen JA, Hansen BF: The value of Nitro BT method in fresh myocardial infarction. Amer heart J. 1973; 85,611-19.
14. Hansen BF: Heart autopsy in ischemic heart disease. An autopsy protocol. Acta Path Microbiol Scand. 1978; 86:241-44.
15. Gregersen N: Myocardial changes in sudden unexpected coronary deaths. Foren Sci. Intl. 1979; 13:183-86.

## **(5) HEART RATE VARIABILITY IN DELAYED SLEEP PHASE SYNDROME**

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## **Abstract**

The study investigated the heart rate variability (HRV) measures and electrocardiogram (ECG) features during sleep after 24 h total sleep deprivation (TSD) in a mild chronic adult male patient of delayed sleep phase syndrome (DSPS). The findings of mean Heart Rate (HR), mean RR (RRI time series), time domain and frequency domain measures are presented. Bradycardias, sinus pauses and ectopic beats were observed during specific parts of sleep. HRVs suggest a sustained sympathetic and decreased parasympathetic activity in the sleep deprived DSPS patient.

## **Keywords**

Delayed sleep phase syndrome, heart rate variability, autonomic nervous system, sleep

## **Introduction**

HRV is an effective non-invasive method providing quantitative evaluation of the autonomic neural activity modulating the cardiac function. It has been widely used to study the sleep associated changes in sympathovagal balance.<sup>1</sup> We present findings of HRV and ECG manual inspection from sleep time after TSD in chronic mild male patient of DSPS. His primary complaint was of frequent sleep onset insomnia episodes (last 2 years) and some issues with sleep maintenance. He is non-alcoholic, smoker (10 years) without suggestive symptoms of anxiety, depression and psychosis. The findings of mean Heart Rate (HR), mean RR (RRI time series), time domain measures of the standard deviation of NN intervals (SDNN) and the square root of the mean squared difference of successive NNs (RMSSD), and frequency domain measures of LF%, HF%, LF nu, HF nu, LF/HF nu are presented.

## **Case report**

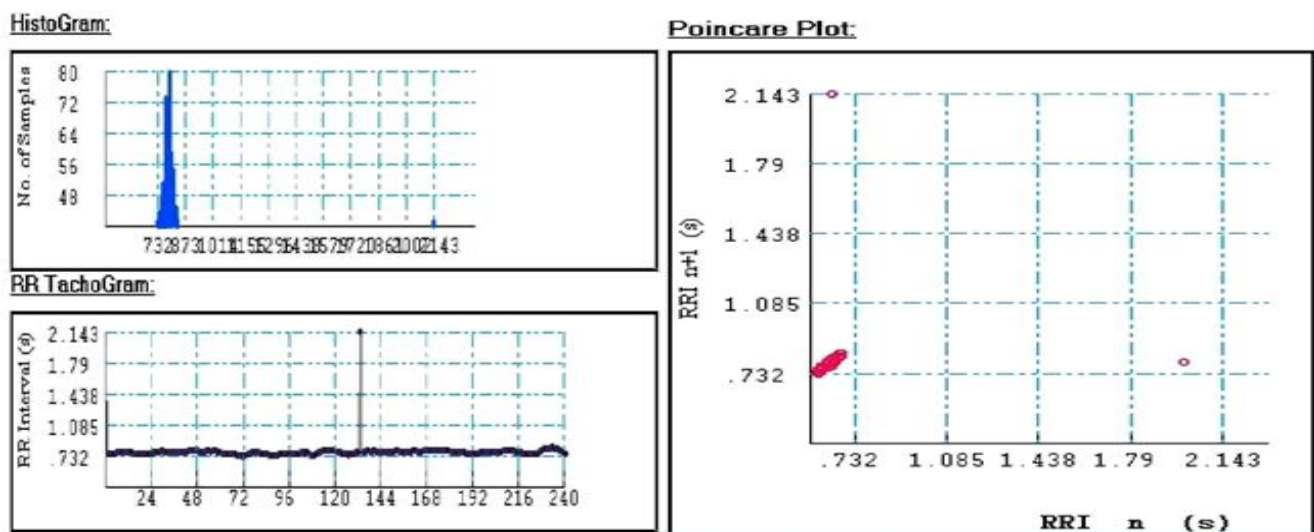
The ECG data for HRV analysis were from the four polysomnographic sleep records performed on a 28 year old Indian male patient of delayed sleep phase syndrome.<sup>2</sup> The sleep records were performed after 24 h total sleep deprivation. The sleep recording time was out of phase with the patients normal sleep time by about 9 h. Single modified ECG lead II comprising torso electrode placement (AASM 2007 manual) was used. Electrodes were placed just beneath the right clavicle and at the midclavicular line (fifth intercostals space) on the left. The HRV analysis was performed using software provided with RMS Quest 32 polysomnograph (Recorders and Medicare Systems, Chandigarh, India). Two records were finally selected after manual

screening for noise, artefacts, ectopic beats and overall quality. The day 2 record was of shorter total sleep time and differed by 1.9 h from day 1. A total of 96 five minute segments of ECG records were evaluated for HR, time and frequency domain measures of HRV. The selection of 5 min intervals and sampling rate of 256 Hz were in agreement with recommendations of the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology.<sup>3</sup> The frequency domain measures were obtained by Lomb Scargle periodogram and Welch's periodogram methods (Hanning window). The Lomb Scargle periodogram is based on Discrete-time Fourier Transform which needs irregularly sampled series sans interpolation requirement. The Welch's periodogram is the traditional Fast Fourier Transform requiring interpolation and even resampling of irregularly sampled series.

For each 5 min segment mean HR, mean RR (RRI time series), time domain measures of SDNN and RMSSD, and frequency domain measures of LF%, HF%, LF nu, HF nu, LF/HF nu were analyzed. For evaluating HRV standard categorization of, LF; 0.04– 0.15 Hz, and HF; 0.15–0.4 Hz was used. All of these measures were grouped according to their sleep stages and their means and standard deviations were calculated. The sleep stage groupings consisted of NREM (stage 1 & 2 Non-rapid eye movement sleep), SWS (stage 3 Non-rapid eye movement sleep), SWS/NREM (segments with stage 3 to stage 1/2 transitions or vice versa), NREM/REM (segments with stage1/2 transitions to REM; rapid eye movement sleep or vice versa) and REM (rapid eye movement sleep). Apart from these, ECG having some special features is discussed.

## Discussion

The manual inspection of ECG revealed some interesting features. Healthy adults have been reported to have a number of cardiac arrhythmias during sleep like bradycardias, sinus pauses, and premature ventricular contractions.<sup>4</sup> It was observed that both the records had more than one sinus pause incidents (RR intervals >2s duration) in SWS and/or SWS/NREM. These were observed near the end of the first SWS episode.



**Figure 1.** It shows the RR tachogram, histogram and Poincare plot of a representative 5 min period with sinus pause.

The RR tachogram, histogram and Poincare plot of a representative 5 min period (with sinus pause) are shown in fig.1. Bradycardias were more frequent in the longer record and occurred

during NREM, SWS and NREM/SWS. The occurrences of ectopic beats were relatively higher in the latter half of both the records. Some of the representative ectopic beats like premature ventricular contractions have been shown in fig.2.



**Figure 2.** The figure shows some of the representative ectopic beats.

**Table 1:** Sleep stage specific summary of the time domain and frequency domain heart rate variables. The frequency domain variables are derived through two algorithms.

The HRV analysis results are summarized in table 1.

	NREM		SWS		SWS/NREM		NREM/REM		REM	
	Day1	Day2	Day1	Day2	Day1	Day2	Day1	Day2	Day1	Day2
HR	65.92±1.93	76.67±2.61	70±1.67	79.77±2.36	67.11±0.93	78.33±3.51	65.00±1.00	75.5±1.73	65.5±0.71	72.5±0.71
RR	0.91±0.03	0.78±0.03	0.86±0.02	0.75±0.02	0.89±0.01	0.77±0.03	0.93±0.02	0.79±0.02	0.92±0.01	0.82±0.01
SDNN	33.88±10.78	34.83±12.57	25.31±5.00	21.47±15.71	28.56±19.76	22.33±2.08	47.6±20.71	45.0±9.69	74±11.31	48±7.07
RMSSD	56.36±5.48	46.32±3.27	52.41±5.03	49.15±18.19	58.90±17.78	44.8±1.30	57.75±2.68	48.49±4.47	56.44±2.62	51.14±6.23
*LF%	16.0±4.89	18.75±6.92	17.5±4.50	18.35±3.92	16.67±4.87	21±1.0	15.2±7.16	11.5±3.42	8.5±0.71	16.5±2.12
*HF%	5.75±3.05	6.25±2.22	6.63±3.20	5.82±3.07	6.33±3.61	5.33±1.16	5.4±2.97	4.0±1.41	3.0±0.0	7.75±1.06
*LF nu	31.1±10.17	31.97±10.39	32.66±6.79	32.37±5.13	33.62±8.59	35.43±4.48	28.14±13.56	19.18±6.18	15.95±3.04	29.7±0.28
*HF nu	11.78±7.98	11.28±5.22	12.88±8.21	11.78±10.09	13.93±10.66	8.77±2.27	9.94±5.51	7.18±2.29	6.1±1.13	10.5±2.83
*LF/HF nu	3.37±1.76	3.83±2.81	3.45±1.84	3.83±1.76	3.33±1.65	4.18±0.83	3.81±2.46	2.74±0.69	2.61±0.01	2.94±0.82
#LF%	5.47±0.64	6.13±0.47	5.74±0.46	6.62±1.11	5.33±0.39	6.87±0.60	5.82±0.95	6.33±0.42	5.75±0.92	5.8±1.41
#HF%	1.45±.20	1.51±0.10	1.48±0.18	1.77±0.95	1.34±0.09	1.53±0.15	1.44±0.11	1.63±0.10	1.45±0.07	1.65±0.50
#LF nu	79.07±2.06	80.23±1.38	79.5±1.86	79.51±3.67	79.97±0.97	82.03±0.32	79.98±2.02	79.58±0.62	79.75±1.77	78.45±0.35
#HF nu	21.00±2.08	19.76±1.40	20.41±2.04	20.34±3.71	20.2±1.27	18.33±0.32	20.02±2.02	20.43±0.62	20.25±1.77	22.15±1.20
#LF/HF nu	3.81±0.43	4.09±0.29	3.93±0.45	4.01±0.59	3.98±0.29	4.48±0.06	4.03±0.49	3.89±0.16	3.96±0.44	3.55±0.21

NREM= stage 1 & 2 Non-rapid eye movement sleep, SWS=stage 3 Non-rapid eye movement sleep, SWS/NREM= epochs with stage 3 to stage 1/2 transitions or vice versa, NREM/REM=epochs with stage1/2 transitions to REM or vice versa; REM= rapid eye movement sleep; HR=heart rate; RR=interval between two R peaks; SDNN=standard deviation of NN intervals; RMSSD=square root of the mean squared difference of successive NN intervals; LF=low frequency (0.04-0.15); HF=high frequency (0.15-0.4); \*Lomb periodogram derived; #FFT linear interpolation derived

All the measures showed marked differences, which were related to sleep stage and day of the record. Probably, this is the first report of HRV measures in DSPS; moreover very little information on the sleep HRV after sleep deprivation is available. The mean HR during both NREM and REM was higher in the patient compared to earlier report in healthy individuals.<sup>5</sup> However, this difference may be because of higher the mean age of the control group and moreover, NREM; had only stage 2 NREM sleep data in the study.<sup>5</sup> The Mean RR during stage NREM, SWS and REM were lower in the patient compared to healthy population of similar age group.<sup>6</sup> The SDNN was relatively very low across all sleep stages compared to reported values in healthy people but SDNN during REM on day 1 was in normal range. Lower overall SDNN has been reported in insomniacs.<sup>5</sup> The RMSSD values were similar on day1; lower on day 2 during NREM and REM vis a vis normal values. The LF % and HF % (LS periodogram) during NREM, SWS and REM was lower than that reported in healthy males but the mean age of volunteers was higher in that study.<sup>1</sup> The LF/HF (LS periodogram) was higher during NREM, SWS and lower during REM compared to healthy males in the literature. Welch's Periodogram derived LF/HF nu were higher in NREM and lower in REM compared to values in literature for healthy controls.<sup>5</sup>

Mean RR and RMSSD were higher in all of the sleep stage groupings in the record with more sleep time. HF nu (both method) were also higher across all sleep but REM (both records) and NREM/REM (Welch's Periodogram). SDNN was higher in longer record in all sleep groupings studied but NREM. Mean HR, LF % (Welch's Periodogram), HF % (Welch's Periodogram) were lower in all stages in longer sleep record, LF% and LF/HF nu (LS Periodogram) also had similar trend except for NREM/REM. LF nu and LF/HF nu (Welch's Periodogram) were lower in stages grouping involving only NREM sleep but higher in those with REM in longer record. LF/HF, LF nu, LF % values normally follow the trend SWS<NREM (stage 1/2) <REM. However, the DSPS patients' data in our case had least values of these measures in REM. This seems to reflect impaired parasympathetic recovery and/or reflect prolonged sympathetic drive.<sup>7</sup> Lower values of HF%, HF nu also lend support to the derivation of decreased parasympathetic activity. Higher mean HR, lower RR and SDNN measures may be related to the sustained sympathetic activity. The elevated HR and decreased HRV have been reported in night workers during rest time suggesting prolonged sympathetic activation and sympathovagal imbalance.<sup>8</sup> DSPS has the characteristic features of circadian disruption and therefore, its involvement in HRV factor changes cannot be ruled out. Moreover, the record timing was also not in alignment with the patients' normal sleeping hours. It would therefore have been more expressive to compare the ECG and HRV characteristics obtained from the normal sleeping hour records.

## References:

1. Elsenbruch S, Harnish MJ, Orr WC. Heart rate variability during waking and sleep in healthy males and females. *Sleep*. 1999; 22(8):1067-71.
2. Manzar MD, Hameed UA, Hussain ME. Total sleep deprivation study in delayed sleep phase syndrome. *Indian J Med Sci*. 2011; 65(4):168-71
3. Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Eur Heart J*. 1996; 17(3):354-81. 2=3

4. Giorgio C. Cardiac arrhythmias during sleep. *Somnologie*. 2000;4: 103-110. 3=4
5. Spiegelhalder K, Fuchs L, Ladwig J, Kyle SD, Nissen C, Voderholzer U, Feige B, Riemann D. Heart rate and heart rate variability in subjectively reported insomnia. *J Sleep Res*. 2011; 20(1.2):137-45. 4=5
6. Busek P, Vankova J, Opavsky J, Salinger J, Nevsimalova S. Spectral analysis of the heart rate variability in sleep. *Physiol Res*. 2005;54 (4):369-76. 5=6
7. Chung MH, Kuo TB, Hsu N, Chu H, Chou KR, Yang CC. Recovery after three-shift work: relation to sleep-related cardiac neuronal regulation in nurses. *Ind. Health*. 2012; 50 (1):24-30. 6=7
8. Su TC, Lin LY, Baker D, Schnall PL, Chen MF, Hwang WC, Chen CF, Wang JD. Elevated blood pressure, decreased heart rate variability and incomplete blood pressure recovery after a 12-hour night shift work. *J Occup Health*. 2008; 50 (5):380-6. 7=8

**(6) EFFECT OF ORAL SILDENAFIL ON RESIDUAL PULMONARY ARTERIAL HYPERTENSION IN PATIENTS FOLLOWING SUCCESSFUL PERCUTANEOUS BALLOON MITRAL VALVULOPLASTY (PBMV): SHORT TERM RESULTS IN 12 PATIENTS.**

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**Introduction:**

Residual pulmonary arterial hypertension is frequently associated with continued symptoms and morbidities of right ventricular (RV) failure even after successful Percutaneous Balloon Mitral Valvuloplasty. (PBMV): Vasodilators and diuretics may further add to the agony of

patients by causing systemic hypotension and hypoperfusion of other organs. Inhaled nitric oxide (NO) and oral sildenafil has been effective in ameliorating symptoms of primary pulmonary hypertension (PPH). Use of oral sildenafil in secondary pulmonary arterial hypertension (PAH) is still investigational. Few favorable reports are noted in recent literature. Our aim is to evaluate the effectiveness of oral sildenafil in patients symptomatic of residual pulmonary arterial hypertension (PAH) by decreasing (RV) after load with maintaining systemic perfusion pressure, oral sildenafil will reduce symptoms and improve effort tolerance in residual PAH patients.

### **Materials and Methods:**

Twelve patients with rheumatic mitral stenosis who underwent successful Percutaneous Balloon Mitral Valvuloplasty (PBMV) in our hospital, but had residual moderate pulmonary hypertension and symptoms related to residual PAH were evaluated for world health organization (WHO) functional class, symptoms level, 6 minute walk test & echo parameters of PAH, Tricuspid Annular Plane Systolic Excursion (TAPSE) and all of them received oral sildenafil 25 mg per day for initial one week followed by 50 mg per day. Patients were followed up in outpatient department with all evaluations repeated at 3 monthly interval including 6 minute walk test and echo parameters. Later, follow up at 6 months was done which included entire work up same as it was done at baseline.

### **Results:**

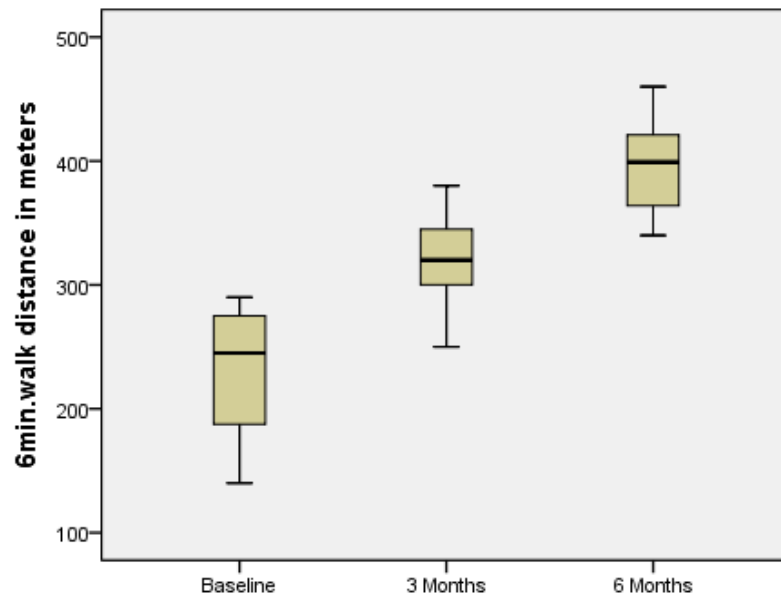
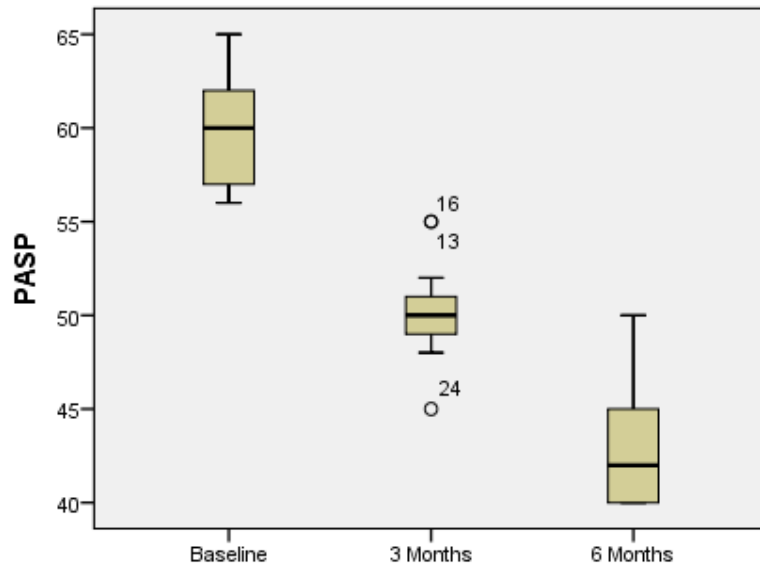
Out of 12 patients who received sildenafil, average time since last procedure of PBMV was 4 months. Seven patients were included in the study with sildenafil at 6 months interval following Percutaneous Balloon Mitral Valvuloplasty (PBMV) procedure. All of them had no improvement in (WHO) functional class after the invasive procedure of Percutaneous Balloon Mitral Valvuloplasty (PBMV). They had moderate PAH and average pulmonary artery systolic pressure (PASP) was 58 mmHg (minimum PASP 56 and maximum values of 65 mmHg.)

Four patients received sildenafil at three months in outpatient follow up after PBMV and one patient started taking the study drug at 7 months after PBMV. Seven patients were recruited while they were in WHO functional class III and five patients were in functional class IV when they started receiving the drug. After 6 months of follow up, WHO functional class improved in all patients with 75% of patients achieving functional class II (N=9) and 25% of patient reached in New York Heart Association (NYHA) class I (N=3). The improvement in functional class was statically significant ( $P=0.002$ ) at 6 months period.

Tricuspid valve Annular Plane Systolic Excursion (TAPSE) was evaluated at baseline and on each follow up. Mean value of TAPSE at baseline was 8.75 mm and it increased to 13.42 mm (46 %) at 6 months follow up ( $P=0.000$ ).

At baseline, patients were made to walk on a straight surface (without slope) for 6 minutes at a stretch. The distance covered in 6 min Walk test was evaluated at each follow up also. Mean distance of 6 min. Walk test was 229.58 meters at baseline which steadily increased to 319.17 meters at 3 months and 395.17 meters at 6 months ( $P=0.05$ ).

## Graphs:



## Discussion:

Moderate to severe mitral stenosis is associated with pulmonary arterial hypertension in the majority of patients (70%). The pathophysiology of PAH in MS has dynamic and fixed components where long standing mitral valve disease induces structural changes in the pulmonary vascular bed mediated by potent vasoconstrictor endothelin-1 (ET-1). Levels of ET-1 are three times higher in severe MS compared with healthy control subjects. Baseline ET-1 concentration has been documented as independent marker that predicts fall in pulmonary capillary wedge pressure (PCWP) at 6 months following PBMV.

Patients with PH secondary to mitral valve disease were not included in clinical trials approved for PAH pharmacologic vasodilator therapies. Few case reports have been published about their

usage in perioperative periods of mitral valve surgery. Agents evaluated are inhaled prostacyclin, inhaled nitric oxide intravenous nitroprusside etc.

Effectiveness use of oral pulmonary vasodilator sildenafil following successful mitral valve surgery with persistent severe PAH has been noted. This novel therapy for PAH is safe and beneficial in residual PAH following PBMV but needs to be validated further in scientifically designed studies.

We aim at reporting initial evaluation of pulmonary vasodilator drug Sildenafil in symptomatic patients with residual moderate PAH following successful PBMV procedure. In the current study sildenafil oral dose 25 mg per day was introduced in significant PAH patients who had successful PBMV and WHO functional class was III and IV. The dose was up-titrated to 50 mg per day after 7 days and there were no side effects or discontinuation of drug and patients were followed up for 6 months following initiation of sildenafil therapy.

Following mitral valve surgery, significant reduction in PASP and pulmonary vascular resistance index (PVRI) is documented in various studies of sildenafil as compared to placebo. Trachte et al (2005) and shim et al (2006) have successfully demonstrated beneficial effects of sildenafil on residual PAH as compared to placebo in patients undergoing mitral valve surgery. This has contributed to rapid weaning from inhaled or intravenous pulmonary vasodilators. Such patients required shorter duration of postoperative ventilatory support. It probably averted the predictable rebound PAH that frequently accompanies weaning from inhaled NO after cardiac surgery. Because sildenafil preferentially inhibits PDE5 which is abundant in lung tissues, it selectively dilates pulmonary vascular beds. We use 50 mg per day dose however it has been demonstrated that peak hemodynamic responses of sildenafil on pulmonary vascular bed are seen in doses as low as 25 mg per day.

In patients with idiopathic primary pulmonary hypertension (PPH) it has been documented that syncope, hemoptysis, decreasing exercise capacity and overt signs of RV failure carry a poorer prognosis. Echocardiographic indices that predict prognostic value are pericardial effusion, indexed right atrium area, LV eccentricity index and tricuspid annular plane systolic excursion (TAPSE). Estimated pulmonary artery systolic pressure (PASP) derived from tricuspid regurgitant jet velocity is not prognostic.

For objective assessment of exercise capacity, the 6 minute walking test is commonly used in patients with PAH. This test is simple, inexpensive, well standardized and easily reproducible. Walking distances  $\leq 332$  meters indicate impaired prognosis in PPH. With respect to treatment effects, good effort tolerance  $\geq 380$  meters following 3 months of IV pulmonary vasodilators correlated well with improved survival in PPH.

Ghofrani HA et al reported beneficial effects of sildenafil in PPH patients and also in secondary pulmonary hypertension with favorable reports on exercise capacity, symptoms and hemodynamics (SUPER-1 study).

We report significant improvement in PASP, TAPSE values and clinical improvements were also seen in WHO functional class as well as distance travelled in 6 minute walk test. All patients exhibited severe symptomatic status in class III and IV at the time of initiation of the study drug and they showed significant benefit and clinical improvement at 6 monthly follow up. It is



noteworthy to state that previous PBMV procedure in the recent past was not effective in demonstrating such an improvement.

Most studies till date have examined efficacy of sildenafil in idiopathic pulmonary arterial hypertension. In secondary pulmonary hypertension, studies have been observational or only case reports, although, they reported benefits with regards to pulmonary and RV hemodynamics.

Our study is also observational, small sample size and needs further evaluation on prospective, randomized study in this subset of residual PAH after successful PBMV. Moreover, whether these improvements in functional capacity, PASP, TAPSE and significant improvement in 6min walk distance are really a response to sildenafil or it is a delayed fall in PASP, slow sustained clinical improvements seen after successful PBMV, the question remains still unanswered. Slow resolution of PAH with delayed improvement may have occurred in some cases. More studies with longer duration of follow up and proper randomization are needed to prove this hypothesis.

### **Conclusion:**

Sildenafil is a drug having several properties as strong pulmonary vasodilator compound. Successful PBMV is expected to resolve PAH in majority of patients. However, few patients continue to exhibit severe symptoms and residual PAH, despite good valve area ( $>1.5 \text{ cm}^2$ ) following PBMV. Such symptomatic patients may exhibit significant improvement after receiving oral sildenafil in follow up period.

Some patients may have chronic obstructive pulmonary disease (COPD) and pulmonary parenchymal disease also. The residual PAH may be attributable to such pulmonary parenchymal disease. Hence, residual PAH after PBMV has some additional contributing causes. Role of oral sildenafil in such cases needs further documentation before its routine use in such cases.

### **References:**

1. Trachte, A. L., E. B. Lobato, et al. (2005). "Oral Sildenafil Reduces Pulmonary Hypertension After Cardiac Surgery." *The Annals of Thoracic Surgery* 2005; 79:194-197.
2. Archer, S. and S. Rich (2000). "Primary Pulmonary Hypertension: A Vascular Biology and Translational Research "Work in Progress"." *Circulation* 2000; 102:2781-2791.
3. Galie N, Ghofrani HA, Torbicki A, Barst RJ, Rubin LJ, Badesch D, Fleming T, Parpia T, Burgess G, Branzi A, Grimminger F, Kurzyna M, Simonneau G, the sildenafil Use in Pulmonary Arterial hypertension (SUPER) study Group. Sildenafil citrate therapy for pulmonary arterial hypertension. *New Engl J Med* 2005; 353:2148-2157.
4. Hopkins WE, Ochoa LL, Richardson GW, et al. "Comparison of the hemodynamics and survival of adults with severe primary pulmonary hypertension or Eisenmenger syndrome." *J Heart Lung Transplant* 1996; 15:100-5.

5. Ghofrani HA, Schermuly RT, Rose F, *et al.* Sildenafil for long-term treatment of nonoperable chronic thromboembolic pulmonary hypertension. *Am J Respir Crit Care Med* 2003; 167: 1139–41
6. Badesch DB, Hill NS, Burgess G, Rubin LJ, Barst RJ, Galie N, Simonneau G. Sildenafil for pulmonary arterial hypertension associated with connective tissue disease. *J Rheumatol* 2007; 34:2417-2422.
7. Ghofrani HA, Olschewski H, Seeger W, Grimminger F. [Sildenafil for treatment of severe pulmonary hypertension and commencing right-heart failure] *Pneumologie* 2002;56:665-672.
8. Prasad S, Wilkinson J, Gatzoulis MA. Sildenafil in primary pulmonary hypertension *N Engl J Med* 2000; 343:1342.
9. G. D. Lewis, R. Shah, K. Shahzad *et al.*, "Sildenafil improves exercise capacity and quality of life in patients with systolic heart failure and secondary pulmonary hypertension," *Circulation* 2007;116: 1555–1562.
10. M. Guazzi, M. Samaja, R. Arena, M. Vicenzi, and M. D. Guazzi, "Long-term use of sildenafil in the therapeutic management of heart failure," *Journal of the American College of Cardiology* 2007; 50: 2136–2144.
11. C. Bomma, H. O. Ventura, G. Daniel, and H. Patel, "Adjunctive sildenafil for the treatment of pulmonary hypertension after mitral valve replacement," *Congestive Heart Failure* 2006; 12: 347–348.
12. J. S. Hung, M. S. Chern, J. J. Wu *et al.*, "Short- term and long-term results of catheter balloon percutaneous transvenous mitral commissurotomy," *American Journal of Cardiology* 1991; 67: 854–862.
13. V. Dev and S. Shrivastava, "Time course of changes in pulmonary vascular resistance and the mechanism of regression of pulmonary arterial hypertension after balloon mitral valvuloplasty," *American Journal of Cardiology* 1991; 67: 439–442.
14. M. C. Walls, N. Cimino, S. F. Bolling, and D. S. Bach, "Persistent pulmonary hypertension after mitral valve surgery: does surgical procedure affect outcome?" *The Journal of Heart Valve Disease* 2008; 17: 1–9.
15. G. Snopek, H. Pogorzelska, T. M. Rywik, A. Browarek, J. Janas, and J. Korewicki, "Usefulness of endothelin-1 concentration in capillary blood in patients with mitral stenosis as a predictor of regression of pulmonary hypertension after mitral valve replacement or valvuloplasty," *American Journal of Cardiology* 2002;90:188-189.

## (7) A COMPARATIVE STUDY OF SIMPLE AUDITORY REACTION TIME BETWEEN MALE CONGENITAL FULL BLIND AND SIGHTED CONTROL

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## **ABSTRACT:**

### **Introduction:**

Huge amount of interest has been created regarding compensatory increase in other sensory modalities in blind due to loss of vision by so called Cross Modal Plasticity theory. In order to review its impact on auditory compensation in blind we attempted to study simple auditory reaction time in male congenital blind and compared with male sighted control's simple auditory reaction time.

### **Methods:**

25 male congenital full blind and 25 male sighted control full filling inclusion criteria were presented with 3000 Hz single tone sound via headphone randomly for 20 times and were instructed to press spacebar as soon as they hear sound. Time interval between sound presentation and pressing of spacebar was recorded by computer using Direct RT software. Other preliminary data were inquired before starting the test.

### **Results:**

Male congenital full blind were having faster reaction time compared to sighted control in significant proportions ( $p < 0.05$ ). Right handed individuals (irrespective of blind or sighted) were having faster reaction time compared to left handed though it was not of significant

magnitude. Negative correlation was found between BMI and reaction time i.e. Higher BMI Lesser simple auditory reaction time

**Conclusion:**

Faster reaction time in congenital blind is proposed to be due to use of occipital cortex for auditory processing due to compensatory reorganization of neuronal circuitry in brain. Though further evaluation of other facets of auditory modality can give clearer picture of this cross modal plasticity.

**KEY WORDS :**

Cross modal plasticity, Handedness, BMI, Auditory reaction time

**INTRODUCTION :**

In 2007, many newspapers all over the world reported that the Belgian police was recruiting blind people as detectives because of their superior auditory skills. One of their jobs has been to analyse tapes of telephone calls; in particular, blind detectives segregated individual voices when listening to a large mixture of sounds and voices of a number of people very precisely. Thus, the blind detectives were better able to infer where a specific suspect was talking or to recognize his/her dialect [1]. When vision is unavailable or insufficient for perception, other sensory modalities often take precedence in sampling the environment. In the case of echolocation used, for example, by many bats and some marine mammals as a mechanism for navigation, object perception, hunting, and social communication [2]. Thus this kind of compensatory neuronal reorganization to enhance remaining sensory modalities is called cross modal plasticity [3].

The reaction time is time interval between application of stimulus and onset of response. Reaction time can be broken down into three parts. The first is perception time: the time for the application and perception of the stimulus and giving the necessary reaction to it. The second is comprehension time for understanding type, duration and direction of stimulus application. The third is motor time, which is the time for compliance to the order received [4, 5]. There are two types of reaction time: auditory and visual. Auditory Reaction time can be described into three types: (1) Simple auditory reaction time - here there is one stimulus and one response. (2) Recognition auditory reaction time - here there are some stimulus that should be responded to and other that should not get response. (3) Choice auditory reaction time - here there are multiple stimulus and multiple responses [6, 7]. John Bernard (1979) described two sensory modalities namely hearing and touch being superior in blind person than sighted. So he carried out research using simple auditory reaction time to compare hearing abilities between blind and sighted individuals [8].

So attempt was made to assess the hearing sensitivities by measuring simple auditory reaction time between blind and sighted persons.

**MATERIALS AND METHODS :**

The study was conducted in Shri Guru Gobind Singh Hospital (tertiary care hospital), Jamnagar, and Gujarat, India. 50 subjects including 25 male congenital full blind and 25 male sighted subjects were included in the study. Prior institutional ethics committee (IEC) approval was taken

before conducting the study. The inclusion criteria were: Age and sex matched subjects from 10 to 50 years old, literate subjects and those who's themselves and/or guardians have given consent were included. Illiterate subjects, person not willing to give consent have been excluded. Subject was either patients, their relatives or visitors to Shri Guru Gobind Singh Hospital, Jamnagar. Subjects were invited for participating in study on voluntary basis. Written consent was taken for participating in study. Auditory tests in form of pure tone audiometry were performed to analyse auditory status of subject. Also reflexes of subject were screened for motor status of subject. A Performa in form of questionnaire contains information about factors affecting reaction time were filled by investigator after interviewing the subject. Reaction time was noted by software Direct RT [9] in milliseconds. Subjects were first explained the procedure. It was carried out in room with adequate light and in silent atmosphere. A 3000 Hz of 90 db was presented randomly through headset provided to them. As soon as person hears the sound he/she has to immediately press the spacebar. Time taken between hearing of sound and pressing of spacebar was recorded in data files. 20 times this exercise was repeated out of which initial 10 reaction time were provided for practice and last 10 reaction times records were included in study. In that 10 reaction time records, minimum and mean simple auditory reaction time records were included. Analysis was done by SPSS version 20.0.0 software. Number of factors thought to be influencing reaction time were analyzed in present study.

## RESULTS :

There were 50 subjects included in the study out of which 25 were male congenital full blind and 25 healthy sighted individuals. In present study age matched male subjects were included in the study in which mean age in male congenital blind is  $23.16 \pm 10.16$  years and sighted is  $23.32 \pm 8.66$  years.

**Table No. - I**  
**Mean ART for Blind and Sighted**

	Groups	N	Mean (mSec)	Std. Deviation (mSec)	p Value
Mean ART	Blind	25	324.52	63.044	0.043
	Sighted	25	367.40	236.241	

**Table-I** shows in present study mean auditory reaction time for congenital full blind was  $324.52 \pm 63.04$  milliseconds while for healthy sighted control it was  $367.40 \pm 236.24$  milliseconds revealing that congenital full blind had faster reaction time than sighted control with data showing significant value  $p < 0.05$ .

**Table No. - II**  
**Minimum ART for Blind and Sighted**

	Groups	N	Mean	Std. Deviation	p Value
Auditory: Mini	Blind	25	261.16	47.522	0.022
	Sighted	25	333.16	148.940	

Considering minimum ART as the fastest response given by an individual, **Table-II** shows that Minimum ART of congenital blind was faster ( $261.16 \pm 47.52$ ) than sighted ( $333.16 \pm 148.94$ ) with significant value  $p < 0.05$

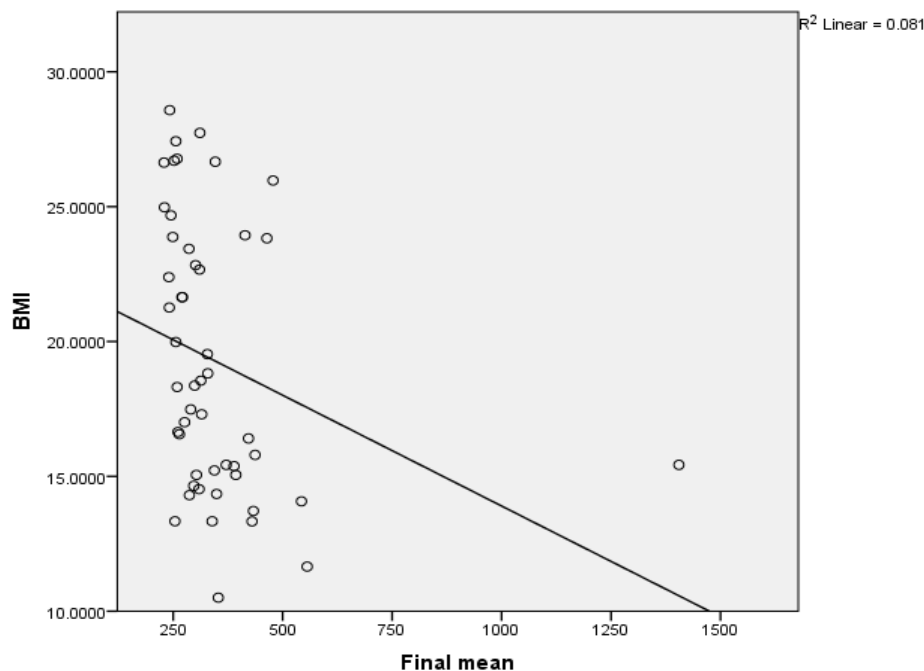
**Table No. - III**

**Comparison of Handedness with mean ART in blind and sighted**

	Hand	N	Mean (mSec)	Std. Deviation (mSec)	p Value
Mean ART	Blind	41	341.54	182.296	0.636
	Sighted	8	374.25	130.551	

**Table-III** shows that out of 50 subjects 41 were right handed individuals and 8 were left handed individuals while 1 person was able to perform various tasks by both hands. Mean auditory reaction time for right handed persons was shorter ( $341.54 \pm 182.3$ ) compare to left handed persons ( $374.25 \pm 130.6$ ). Though results were found to be insignificant inspite of difference in mean ART ( $p > 0.05$ ).

Considering Body Mass Index (BMI) of study group, mean BMI of study group was  $19.28 \pm 4.99$ . Correlation coefficient study for observing effect of BMI on mean ART showed negative ( $-0.284$ ) correlation i.e. MORE BMI LESS MEAN ART with  $R^2 = 0.081$ . See Graph-I.



**GRAPH NO. – I :** Correlation of BMI with mean ART

BMI= sq.m/kg, Final mean ART= millisecond

### **DISCUSSION:**

Study conducted by Collignon O et al [10] shows that blind have exceptional abilities in auditory processing due to recruitment of occipital areas for auditory processing. Another study done by

Kirsten Hotting et al [1] showed that increase use of auditory system results in compensatory behavior having better perceptual auditory tasks. Mario Liotti et al [11] group showed that reaction times for attend ear intensity deviant targets were markedly faster (91msec faster) for blind compare to sighted individuals. While O. Despres et al [12] group results were showing that early blind subjects exhibited shorter reaction time than sighted when sound sources were placed at far lateral locations. Thus supporting results of present study that simple ART of congenital full blind is faster (43 msec faster) than sighted (See Table-I). Though study done by John Bernard [7] showed insignificant reaction time results between blind and sighted individuals. Different activation patterns in the visual cortex of late and congenitally blind subjects was also shown by Christian Buchel et al [13]. Animals and humans that grow up blind use their auditory modality for localization in far space. Areas in parietal and occipital cortex that are ordinarily used for vision become activated by hearing input. This leads to an expansion of auditory areas in the dorsal stream into visual area and to a simultaneous sharpening of auditory spatial tuning in these neurons. Together, this massive cross-modal reorganization leads to superior performance of blind as compared to sighted individuals in auditory spatial tasks. [14]

The right hemisphere controls the left hand, and the left hemisphere controls the right hand. Sathiamoorthy A et al [15] study showed that left handed volunteers were having faster reaction time compare to right handed volunteers. Contrary to that in present study right handed individuals were having faster reaction time compare to left handed individuals using dominant hand. Study conducted by Lalita H Nakim and Jayshree V. Gadkari [16] showed that there is positive correlation of BMI with reaction time which is contradictory to this study.

### **CONCLUSION:**

Considering the ability of brain neurons to reorganize among themselves to enhance the residual sensory modalities can be utilized in increasing brain performance by reversible induced suppression of one specific sensory modality. So we conclude that in present study congenital full blind have faster ART compare to sighted control. Though further evaluation of various aspects of sensory modalities in blind along with elaborate research on factors affecting them can enlighten mechanism underlying neuronal reorganization in blind subjects.

### **ACKNOWLEDGEMENTS:**

We are thankful to ENT department, Physiology department, Guru Gobind Singh Hospital & Shree M.P Shah medical college, Jamnagar & volunteers for their heartily cooperation.

**CONFLICT OF INTEREST:** None declared

### **REFERENCES:**

- (1) Kirsten Hotting, Brigitte Roder (2009). Auditory and auditory-tactile processing in congenitally blind humans. *Hearing Research* 258 (2009) 165–174
- (2) Thomas JA, Moss CF, Vater M (2004) *Echolocation in bats and dolphins*. University of Chicago Press, Chicago 631 pages | 10 halftones, 285 line drawings, 15 tables | 8-1/2 x 11 | © 2003

- (3) Bavelier, D., Neville, H.J., 2002. Cross-modal plasticity: where and how? *Nat. Rev. Neurosci.* 3, 443–452
- (4) Teichner WH (1954). Recent studies of simple reaction time. *Psychol Bull* 51:128
- (5) Tripo RS (1965). How fast can you react? *Sci. Dig* 57:50
- (6) Luce RD, *Information Theory of Choice-Reaction Times*. London: Academic Press; 1968. Available from <http://biology.clemson.edu/bpc/bp/Lab/110/reaction.htm>. (last accessed on 08/08/2011)
- (7) Welford, AT. 1980. Choice reaction time: Basic concepts. In A. T. Welford (Ed.), *Reaction Times*. Academic Press, New York, pp. 73–128
- (8) John Bernard (1979). Simple auditory reaction time in blind and sighted adolescents. *Percept Mot Skills*. 1979 Apr;48 (2):465-6
- (9) Website: <http://www.empirisoft.com/directrt.aspx> as sited on 21/03/2013
- (10) Collignon O, Voss P, Lassonde M, Lepore F. (2009). Cross-modal plasticity for the spatial processing of sounds in visually deprived subjects. *Exp Brain Res*. 2009 Jan; 192(3):343- 58. Epub 2008 Sep 2
- (11) Mario Liotti, 2, CA Kathy Ryder' and Marty G. Woldorff 1,2. (1998). Auditory attention in the congenitally blind: where, when and what gets reorganized?. *Neuro Report* 9, 1007- 1012 (1998)
- (12) O. Despres, V. Candas, A. Dufour (2005). Spatial auditory compensation in early-blind humans: Involvement of eye movements and/or attention orienting? *Neuropsychologia* Volume 43, Issue 13, 2005, Pages 1955–1962
- (13) Buchel, C., Price, C., Frackowiak, R.S., Friston, K., 1998. Different activation patterns in the visual cortex of late and congenitally blind subjects. *Brain* 121, 409–419
- (14) Website: [http://link.springer.com/chapter/10.1007/978-0-387-71978-8\\_20](http://link.springer.com/chapter/10.1007/978-0-387-71978-8_20) as sited on 21/03/2013
- (15) Sathiamoorthy A, Sathiamoorthy SS, Bhat SK, Hiremath S, Shenoy N Influence of handedness on the visual and auditory reaction time. *Indian J Physiol. Pharmacol* 1994 Oct;38(4):297-9.
- (16) Lalita H. Nikam, Jayshree V. Gadkari (2012). Effect of age, gender and body mass index on visual and auditory reaction time in indian population. *Indian J Physiol Pharmacol* 2012; 56(1): 94–99



## Original Article

**(8) MICROALBUMINURIA : A MARKER OF CRITICALLY ILL PATIENTS**

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**ABSTRACT****INTRODUCTION:**

In Critical care units, prediction of outcome is of vital importance to the clinician. It allows planning of early therapeutic intervention and appropriate counseling of patient. Prognostic measures used in ICU should ideally detect short term changes in critical illnesses and also reflect impact of early therapeutic interventions on the outcome of patient. So sensitive, inexpensive and dynamic prognostic markers which generate rapid and reliable results are therefore desirable in the ICU setting.

**OBJECTIVES:**

The goal of our study is to evaluate Microalbumin to Creatinine ratio within 6 hours of ICU admission (ACR1) and after 24 hours (ACR2) of ICU admission and to correlate Microalbumin to Creatinine ratio with the APACHE-II score to predict outcome in critically ill patients.

**METHOD:**

In this prospective non-interventional study, 100 adult patients admitted to M.I.C.U and S.I.C.U with more than 24 hours of ICU stay were included and after their informed consent, blood and urine samples were collected on admission to ICU and 24 hours thereafter. Urine microalbumin to creatinine ratio (ACR1) was measured on admission and after 24 hours of ICU admission. For disease severity scoring, APACHE II scores were calculated.

**OBSERVATION AND RESULT:**

Out of 100 patients, 82 survived while 18 patients died in ICU. Non-survivors had significantly higher median ACR2 [164.5 mg/g (IQR 104.9-172.1)] in comparison to the survivors who had a median ACR2 [46.0 mg/g (IQR 25.6-89.4)] ( $P < 0.0001$ ). The median ACR1 [161.5 mg/g (IQR 105.3-180.8)] of non-survivors was also significantly higher than median ACR1 of survivors [89.8 mg/g (IQR 28.7-101.3)] ( $P < 0.0001$ ). In a receiver operating characteristic curve (ROC) analysis, ACR2 merged as the best indicator of mortality [area under curve (AUC) of ACR2 = 0.85 > AUC of ACR1 = 0.81 > AUC of  $\Delta$ ACR = 0.61] similar to the currently used APACHE II scores (AUC = 0.90). At a cut off of 101 mg/g, ACR2 had sensitivity of 83.3%, specificity 79.7%, positive predictive value of 48.3% and negative predictive value of 95.4% for predicting mortality in critically ill patients.

**CONCLUSION:**

Microalbuminuria at 24 hrs of ICU admission had a good sensitivity and specificity to predict mortality equivalent to APACHE II scores and could be effectively utilized to predict survival in the ICU.

**KEY WORDS:** Critically ill, intensive care unit, Microalbuminuria, mortality, outcome

**INTRODUCTION**

The development of Intensive Care Unit (ICU) has increased the ability to monitor, diagnose and treat critically ill patients (1). Predicting patients outcome is an important component of patients care in critical care units. (2). It allows planning of early therapeutic interventions, optimal resource allocation and appropriate counseling of the family and/or patient. The two widely used adopted systems to predict mortality are the Acute Physiology and Chronic Health Evaluation (APACHE-II) and the Simplified Acute Physiology Score (SAP-II) scores. Though useful to evaluate outcome, these cumbersome tools are of limited use in day to day practice. Prognostic measures employed in the ICU should ideally detect short term changes in critical illnesses and also should reflect the impact of early therapeutic interventions on the outcome of the patient. Sensitive, inexpensive and dynamic markers which generate rapid and reliable results are therefore desirable in the ICU setting. Critical illnesses are more often characterised by the Systemic Inflammatory Response Syndrome (SIRS), the host response to an acute insult. SIRS is a common finding in the ICU patients, which when severe can lead to Multiple System Organ failure (MSOF) and finally death. A severe and sustained inflammatory reaction induces rapid and profound changes in the endothelium resulting in the loss of barrier integrity leading to systemic capillary leak. In the Kidneys these manifests as altered glomerular permeability culminating in increased renal albumin excretion in the urine (3). Such low rates albumin excretion (<300 mg/day) are termed Microalbuminuria. The degree of microalbuminuria can be reliable index with a spot urine albumin-creatinine ratio (ACR) (4). Microalbuminuria is used clinically to monitor incipient Diabetic Nephropathy, but it is known to be useful as a predictor of outcome in several clinical situations such as Acute Myocardial Infarction, Cerebral Ischemia, Ischemic Heart Disease, Burns, Meningitis and Ischemic Reperfusion Injury. In such conditions, Microalbuminuria has rapid onset and typically lasts for <48 hours unless complications occur (2, 5, 6).

## AIM & OBJECTIVES

Thus this study was undertaken to infer whether the degree of microalbuminuria after 24 hours of ICU admission would reflect the degree of ongoing endothelial dysfunction after goal-directed therapy and it could predict mortality in critically ill patients better than microalbuminuria at admission as it would reflect the host response to early therapeutic intervention.

## MATERIAL & METHODS

The present study was conducted at Shree Sayaji Rao General Hospital and Medical College, Vadodara after obtaining approval from the institutional ethical committee. The subjects for the study included 100 adult patients admitted to M.I.C.U. and S.I.C.U. and 100 healthy subjects taken from medical or paramedical staff and general population. After taking informed consent, all adult patients >18 years old with a ICU stay more than 24 hours were included.

### **The patients with the following diagnosis were excluded from the study:**

Anuria, Macroscopic Hematuria, Preexisting Chronic Kidney Disease, Female Patients with Menstruation or Pregnancy, Patients with significant proteinuria (more than 1 + protein on dipstick) due to renal and post renal causes eg. Urinary Tract Infection.

APACHE - II scores were calculated from data collected during the first 24 hrs following ICU admission.

All patients had Foley's Urinary catheter in place. Spot Urine samples were collected in sterile container without preservative. Microalbumin was done after screening the samples by Uristix for any protein. Negative samples (negative/trace) by Uristix were estimated for Microalbumin. Urine was collected within 6 hrs of admission and again at 24 hrs for quantification of urine microalbumin to creatinine ratio which was referred to as ACR 1 and ACR 2 respectively.

Urine Microalbumin was determined by Pyrogallol Red method manufactured by Crest biosystems and done on semiautomated Biochemistry analyzer ERBA CHEM-5 plus and Urine Creatinine by Modified Jaffe's Kinetic Method manufactured by Reckon diagnostics done on semiautomated Biochemistry analyzer ERBA CHEM-5 plus. Microalbuminuria was expressed as ACR (Albumin to creatinine ratio) values between 30 to 299 mg/g. ACR of >300mg/g is considered as clinical proteinuria. ACR <30 mg/g is normal for a healthy population. Trend of microalbuminuria was assessed from the change of ACR value within 6 hours of admission (ACR1) to the ACR value at 24 hrs (ACR2) in both groups of patients. The difference of ACR2 from ACR1 ( $\Delta\text{ACR} = \text{ACR1} - \text{ACR2}$ ) was calculated.

## STATISTICAL ANALYSIS

A statistical analysis was done by using Kolmogorov - Smirnov test to assess sample distributions. The results are presented as the median and 25th / 75th percentile (Inter quartile range, IQR). [ACR (Microalbumin to Creatinine ratio) data non normally distributed]. To compare two independent samples we used the Mann-Whitney U test (data non-normally distributed). P value <0.05 was considered significant. Receiver operating characteristic (ROC) curves and the area under the ROC curves were calculated for ACR1, ACR2,  $\Delta\text{ACR}$  and the APACHE II score. The discriminating power of ACR variable (ACR1, ACR2,  $\Delta\text{ACR}$ ) for the prediction of mortality was

assessed by the area under the ROC curve. From the ROC curves diagnostic accuracy of the variables were determined by calculating their respective area under the curve (AUC).

## RESULTS

Patient demography, medical/surgical classification, number of patients with diabetes and hypertension, median duration of ICU stay, number of survivors and non-survivors in Group II are summarised in Table 1.

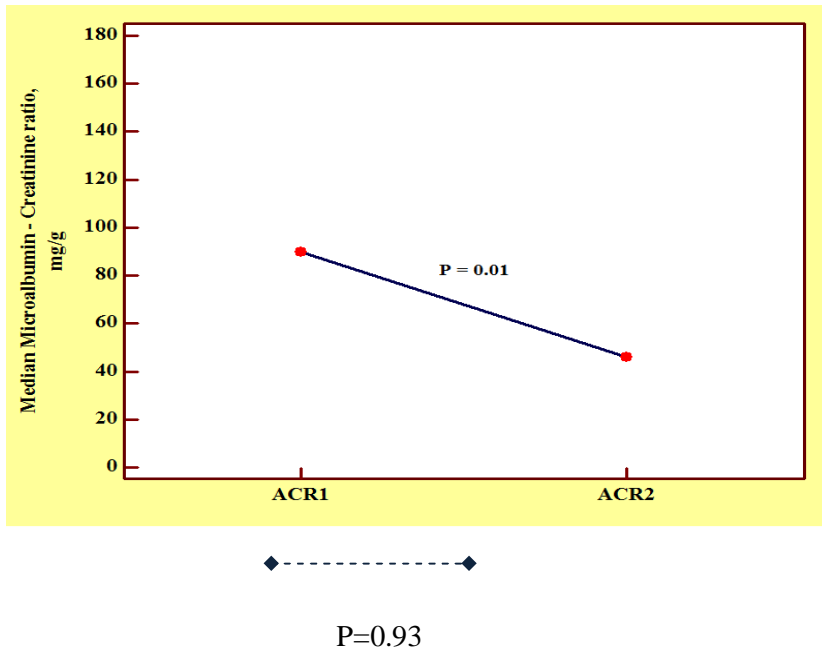
**Table 1: Baseline characteristics of Group II (CASES)**

No. of patients	100
Median age, years (IQR)	52 (41.5- 65 )
Male (%): Female (%)	53 % : 47 %
Medical (%): Surgical	95 % : 5%
Patients with diabetes mellitus (%)	37 %
Patients with hypertension (%)	33 %
Median duration of ICU stay, days (IQR)	4(3 – 5)
Survivors (%)	82 %
Non-Survivors (%)	18 %

On admission, 29 patients had ACR < 30 mg/g, 71 patients had microalbuminuria (ACR between 30-300 mg/g). At 24 hours of admission, 32 patients had ACR < 30mg/g, 68 patients had microalbuminuria (ACR between 30-300 mg/g).

A comparison between the survivors and non-survivors showed that the non-survivors had a significantly higher median APACHE II score [35(IQR 28-40)] and median duration of ICU stay [6 (IQR 3-10)] as expected than survivors who had median APACHE II score [18(IQR 12-22)] and median duration of ICU stay [5(IQR 3-7)] ( $P < 0.0001$ ) and ( $P < 0.002$ ) respectively. Median ACR<sub>2</sub> were significantly higher in the patients who died in the ICU [164.5 (IQR 104.9-172.1) mg/g in comparison to those who survived [46.0(IQR 25.6-89.4)] mg/g ( $P < 0.0001$ ). Median ACR<sub>1</sub> was also higher significantly higher in non-survivors [161.5(IQR 105.3-180.8)] mg/g than survivors [89.8(IQR 28.7-101.3) mg/g  $P < 0.001$ . There was no significant difference in  $\Delta$ ACR between survivors [12.3(IQR -7.6-26.8)] mg/g and non survivors 3.0 (IQR -11.1-10.0)] mg/g ( $P = 0.12$ ).

In survivors there was a significant decrease in the median level of ACR 1 [89.8(IQR 28.7-101.3)] mg/g to the median level of ACR 2 [46.0(IQR 25.6-89.4)] mg/g ( $P = 0.01$ ). (Fig 1)



**Fig 1: Change in Median Microalbumin to creatinine ratio at ICU admission (ACR 1) and after 24 hours (ACR 2) in survivors And non-survivors (●—● survivors, ◆-----◆ non-survivors)**

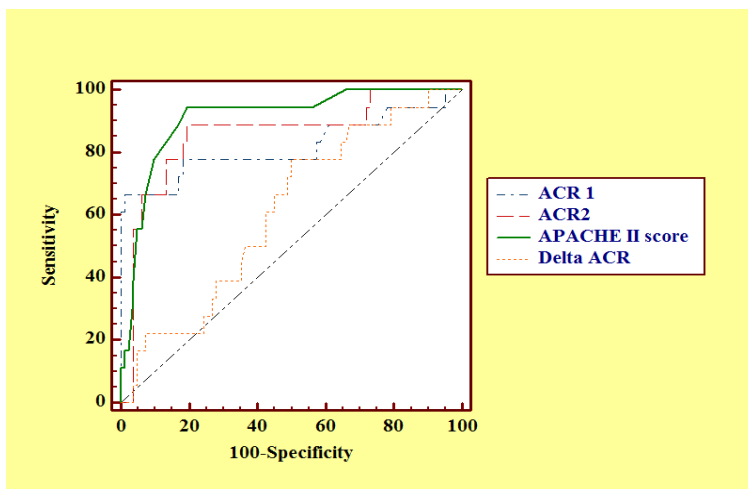
However in non-survivors there was no significant decrease in median ACR [161.5(IQR105.3-180.8)] mg/g to the median ACR 2 [164.6(IQR 104.0-172.1)] mg/g. (P=0.93). (Fig 1)

Also, ACR1, ACR2 and Δ ACR strongly correlated with APACHE II scores (Table 2)

**Table 2: Correlation of APACHE II score with Urine Albumin**

	ACR1	ACR2	Δ ACR
APACHE II Score	P<0.0001 rs=0.6	P<0.0001 rs=0.85	P=0.04 rs=0.19

For the entire population, the area under the ROC curves for mortality was highest for APACHE II Score (Area under curve 0.90), then ACR2 (0.85), followed by ACR1 (0.81), and Δ ACR (0.61). There was no significant difference between the area under the curve for APACHE II and ACR2 (P=0.12). (Fig 2)



## Fig 2: Comparison of ROC curves of APACHE II, ACR1, ACR2 and $\Delta$ ACR

To estimate the diagnostic accuracy of the urine ACR in the prediction of mortality, the sensitivity and specificity were determined using the conventional cut-off level of microalbuminuria at 30mg/g. At this value, ACR2 had a sensitivity of 89% with a negative predictive value of 94% for the prediction of ICU death. However, the positive predictive value of death was 23%. To explore a wider clinical utility of the ACR test to include a larger subset of population, the cut-off was increased to 101 mg/dl. At this cut-off, the negative predictive value was 95%. However the positive predictive value was 48%. (Table 3)

**Table 3: Sensitivity, specificity, positive predictive value and negative predictive value of ACR 2 at two different cut off values for prediction of mortality**

ACR 2 Cut off mg/g	Sensitivity %	Specificity %	Positive predictive value of death %	Negative predictive value of death %	Percentage of patients below cut off
≤ 30	88.9	35.3	23.1	93.5	31
≤ 101	83.3	79.7	48.3	95.4	66

## DISCUSSION

In the present study, of all the patients admitted to ICU, microalbuminuria was found to be prevalent in broad spectrum of critically ill patients. 71% patients had ACR>30 mg/g on ICU admission and it persisted in 68% at 24 hours of ICU admission. At 24 hours, 34% of patients had ACR levels of more than 101 mg/g.

S. Basu et.al (3) in their study of 238 critically ill patients, found that on admission 76% of patients had ACR >30mg/g on ICU admission and it persisted in 67% at 24 hours. At 24 hours, 43% of patients had ACR levels of more than 101mg/g.

Microalbuminuria in critically ill patients is probably the result of widespread endothelial dysfunction arising from the effects of cytokines and other inflammatory responses that are associated with critical illnesses. The cascade of events triggered by the release of interleukins and other inflammatory mediator's results in widespread endothelial dysfunction. The effects of disruption of the integrity of the endothelial barrier is manifested as altered glomerular endothelial permeability in the kidneys, allowing increased amounts of albumin to escape into the glomerular ultrafiltrate. The tubular resorptive is exceeded beyond its threshold capacity, leading to increased excretion of albumin in the urine. The degree of microalbuminuria is dependent on the intensity of inflammatory responses and therefore microalbuminuria reflects disease severity (3).

In the present study, we found that non-survivors had higher levels of microalbuminuria on ICU admission compared to survivors. Also there was significant increase in microalbuminuria in non-survivors at 24 hrs. On the contrary, the median ACR2 fell significantly in survivors. This finding corroborates with previous studies of Mackinnon et al (7), S. Basu et al (3), Gosling et al (8), and Omar Abid et al (6). This finding indicates that failure of ACR to decline is associated with increased ICU mortality.

In our study, ROC curve analysis revealed that measurement of ACR at 24 hrs of ICU admission was a better predictor of death compared to that on admission and fared as well as the time-tested APACHE-II scores. ACR2 at a cut-off of 30 mg/g had a good negative predictive value (93%). Study by S. Basu et al (3) also found that at a cut-off of 30mg/g ACR2 had negative predictive value of 94% for prediction of mortality.

Our study had few limitations. Firstly our study was predominantly medical (>90%) and so it would not be appropriate to extrapolate the results to a surgical ICU population immediately. Secondly patients with diabetes and hypertension were included which are independent causes of microalbuminuria, since their exclusion would have made the study population less representative of real life scenario. Further studies could be directed towards evaluating the use of microalbuminuria in monitoring the efficacy of individual therapeutic interventions which impact survival, in critically ill patients.

### CONCLUSION

Microalbuminuria at 24 hrs of ICU admission had a good sensitivity and specificity to predict mortality equivalent to the time-tested APACHE II scores. The study indicated that microalbuminuria is an inexpensive, rapid diagnostic tool & could be effectively utilised to identify patients likely to survive in the ICU.

### REFERENCES

1. Asodalah Saadat-Niaki, Dariush Abtahi. Predicting the Risk of Death in Patients in Intensive Care Unit. Arch Iranian Med.2007; 10(3): 321-326.
2. Basim A Dubaybo. Microalbuminuria: Simple, Inexpensive and Dynamic Marker of Critical Illness. Chest 2001; 120(6): 1769-71.
3. S. Basu, S. Chaudhari, M. Bhattacharya et al. Microalbuminuria: An Inexpensive, Non Invasive Bedside tool to predict outcome in Critically Ill patients. Ind J of Clin Biochem 2010; 25(2): 146-152.
4. Azim H, Mohammadreza S, Kevian B. et al. The association of microalbuminuria and duration of mechanical ventilation in critically ill trauma patients. Tur J of Trauma & Emergency Surgery 2009; 12-18.
5. Evans G, Greaves I. Microalbuminuria as a predictor of outcome shows promise but large prospective trials are needed. BMJ 1999; 318(7178): 207-8.
6. Abid O. Sun Q, Sugimotak et al. Predictive value of microalbuminuria in medical ICU patients: Results of a pilot study. Chest 2001; 120: 1984-8.
7. K. L. Mackinnon, Z. Molnar, D. Lowe et al. Use of microalbuminuria as a predictor of outcome in critically ill patients. Br J of Anaesth. 2000; 84(2): 239-41.
8. Gosling P. et al. Microalbuminuria in intensive care unit: Clinical correlates and association with outcomes in 431 patients. Crit Care Med 2006; 34(8): 2158-66.

## **(9) ASSOCIATION OF RISK FACTORS WITH SEVERITY OF DIABETIC RETINOPATHY IN HOSPITAL BASED POPULATION - SAMVEDANA EYE CARE FOR DIABETES – A PROJECT OF NAGRI EYE HOSPITAL**

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### **PURPOSE:**

To study the effect of additional risk factors on severity of Diabetic Retinopathy in Hospital based Diabetic population.

### **METHOD:**

In this hospital based study screening of 1,48,343 patients from 1<sup>st</sup> January 2007 to 28<sup>th</sup> Feb. 2013 was carried out. Out of the total screened 36971 were found to be Diabetes positive. These patients were then subjected to detailed medical & ophthalmological check up following which they were sub-grouped into categories with specific additional risk factor.

### **RESULTS:**

Of all the risk factors five main associations namely Hypertension, Ischemic Heart Disease, Hypertension with Ischemic Heart Disease, Nephropathy & Neuropathy were found to Be most prevalent. Of the total Diabetes Mellitus positive patients 5735 had diabetic retinopathy.

### **CONCLUSION:**

Of all the important risk factors diabetes alone and Hypertension with co-existent Diabetes were found to be most prevalent and hence most significant. Cases of co-existent Nephropathy shown to have more severity of disease but our Outpatient Data was insufficient as most of the time patients were not aware of the problem.

### **INTRODUCTION**

DR is one of the main causes of visual loss in individuals aged 20-64 years old and is present in more than 77% of patients with DM type 2 having disease duration of more than 20 years.<sup>1,5</sup> Visual impairment and registered blindness due to diabetic retinopathy accounts for 17.6% and 0.2% of the cases, respectively.<sup>2</sup>

Many factors have been associated with the progression and severity of DR, such as DM duration<sup>3,5</sup>, the control of serum hyperglycaemia<sup>4,5,6</sup> and hypertension<sup>5,6,7</sup>. The UKPDS trial<sup>8</sup> is one of the largest trial done to understand the effect of additional risk factors on



development of DR. It has shown that chronic hyperglycemia and hypertension are the most important risk factors. It also showed that DR occurs in approximately 84.5% of patients after 15 years of the disease<sup>8,9</sup>.

## **BACKGROUND**

Early detection of diabetic retinopathy and treatment with laser therapy can prevent visual impairment and most patients can be saved from major visual loss<sup>10</sup>

To meet this challenging health-economic burden of society, a first ever project in Gujarat: SAMVEDANA EYE CARE FOR DIABETES has been initiated by Nagri Eye Research Foundation in October 2006. Samvedana, a USA based trust, joined the project in January 2007.

Subsequently, World Diabetes Foundation, Denmark has appraised the project, and is now providing much needed financial and technical aid.

In this study, we aimed to investigate the independent associations between the stage of DR and a variety of risk factors, like hyperglycemia, hypertension, diabetic neuropathy, diabetic nephropathy and ischemic heart disease in samvedana eye care for diabetes project.

## **MATERIALS & METHODOLOGY:**

This was a hospital based Our Diabetic retinopathy screening program included a team of an ophthalmologist, optometrist, Lab. Technician, community health workers and a health counselor.

Screening programs included 3 teams. The city of Ahmedabad was divided into 5 different zones. Screening of diabetic patients was done at different zonal UHC. One team was visited various urban health centers of Ahmedabad city. One team visited the 3 general hospital of Ahmedabad city on periodic intervals and one team was always based at the C. H. Nagri municipal eye hospital.

All the patients above the age of 40 participating in the study underwent complete. Evaluation Detailed history including personal data like name, age, was taken. Detailed systemic history was elicited including the type of diabetes, its duration, whether patient is on oral treatment or insulin, and whether having associated systemic illness like hypertension or hypercholesterolemia and its duration and control. Associated history of complications of diabetes in other organs like nephropathy and neuropathy was elicited.

Detailed ophthalmic examination was done including best corrected visual acuity (BCVA) measurement, IOP measurement by Schiotz tonometry and slit lamp biomicroscopy. A complete retinal assessment with a 90 diopter fundus lens and Binocular indirect ophthalmoscopy was done. DR was clinically graded in accordance with the Early treatment diabetic retinopathy study (ETDRS) guidelines<sup>10</sup>. They were - Non proliferative diabetic retinopathy, proliferative diabetic retinopathy, very severe proliferative diabetic retinopathy and clinically significant macular edema.

Those having diabetic retinopathy were counseled regarding further treatment and also referred to a diabetologist to control their systemic factors.

**RESULTS :****Table: 1 Baseline data**

Total no. of patients screened	Diabetes Positive	Diabetes Negative	Diabetic retinopathy
1,48,343	36971	111,372	5735

**Table: 2 Variables**

Variables	
Age	54.67 ±9.4 yrs
Random Blood Sugar (mean)	235.6 ± 43.5 mg/dl
Mean duration of diabetes	12 ±5.8 yrs
Diabetic retinopathy with Hypertension	20.03%
Diabetic retinopathy with Ischemic Heart Disease	15.22%
Diabetic retinopathy with Neuropathy	8.12%
Diabetic retinopathy with Nephropathy:	10.82%
Diabetic retinopathy with diabetes alone	45.78%

**Table: 3 Association of risk factors with type of diabetic retinopathy**

	NPDR (3720)	PDR (479)	SEVERE PDR (99)	CSME (1437)
Diabetes + Hypertension (1149) (20.03%)	14.33%	1.62%	0.27%	3.80%
Diabetes + Ischemic heart disease (873) 15.22%	11.35%	0.94%	0.73%	2.59%
Diabetes + Nephropathy (621) 10.82%	6.67%	1.37%	0.38%	2.4%
Diabetes + Neuropathy (466) 8.12%	5.9%	0.73%	0.069%	1.41%
Diabetes (2626) 45.78%	26.59%	3.67%	0.66%	14.85%

**DISCUSSION :**

Diabetes is a chronic disease with micro and macro vascular complications. Diabetic retinopathy is the most common micro vascular complication of diabetes. In our study out of the total of 148,343 people 36971 were having diabetes out of which 5735 were found to have diabetic retinopathy.

Multiple studies have given the risk factors for development of diabetic retinopathy<sup>5,6,8,9</sup>. Klein et al in their study suggested that chronic uncontrolled diabetes is the most important risk factor for development of diabetic retinopathy. In our study the mean duration of diabetes was 12 yrs with mean random blood sugar of 235mg/dl suggesting a chronic uncontrolled disease among the study population. In our study diabetes was the most important risk factor, associated with higher risk of all the types of diabetic retinopathy.

Multiple other studies have shown hypertension as the second most common risk factor. In our study 20.03% of the population with diabetic retinopathy had hypertension. Hypertension was most significantly associated with Non proliferative type of diabetic retinopathy.

Stratton et al. assessed the effects of glycemia and systolic blood pressure exposures on the risk of diabetic complications over a median of 10.4 years. They reported risk reductions of micro vascular complications, including retinopathy of 21% per 1% glycosylated hemoglobin A1c decrement and 11% per 10 mmHg systolic blood pressure decrement and concluded that intensive treatment of both of these risk factors is needed to substantially reduce the incidence of these complications.

Third risk factor in our study was ischemic heart disease. It was present in 15% of the study population and was present in 19% of the high risk proliferative diabetic retinopathy suggesting a more important correlation ship between macro vascular complication of diabetes.

Diabetic nephropathy was found in 10% of the study population and it was found in higher % among the population having proliferative (17.5%) and high risk proliferative diabetic retinopathy (19.8% of total PDR patients).

Diabetic nephropathy represents a major micro vascular complication of chronic diabetes and multiple studies have already proven the correlation between diabetic nephropathy and diabetic retinopathy<sup>11,12</sup>. However, our outpatient data was not sufficient to rule out nephropathy as most of the patients were not aware of their condition and if patient's medical records were available may be a stronger association could have been found between diabetic retinopathy and nephropathy.

DR is the most common and preventable cause of vision loss in adults in developed as well as the developing countries and the prevalence of both diabetes and diabetic retinopathy are expected to increase exponentially in the next 30 years<sup>1,2,7</sup>.

As the incidence of Diabetes increases, it will be utmost important for clinicians who care for patients with diabetes to be aware of the risk factors associated with diabetic retinopathy. Identifying risk factors for DR in patients with diabetes may help to identify a subgroup of patients with higher priority for DR screening and improve early screening for those at highest risk.

Vision loss from DR can be avoided by early detection through annual ocular screenings and earlier recognition of diabetes, better glycemic control, and early detection and treatment of DR

### **CONCLUSION :**

This is the first of its kind hospital based diabetic retinopathy screening project where screening of more than 1, 00,000 patients in western Indian urban areas has been done.

In our study diabetes alone and Hypertension with concomitant diabetes were found to be the most common risk factor associated with diabetes. This study will help the physicians to identify patients who are at higher risk of developing vision threatening diabetic retinopathy and will in turn help in prevention of this grave Blinding condition.

### **REFERENCES :**

1. Centers for Disease Control and Prevention (CDC) Blindness caused by diabetes-- Massachusetts, 1987-1994. MMWR Morb Mortal Wkly Rep. 1996;45:937-941.

2. Bamashmus MA, Gunaid AA, Khandekar RB. Diabetic retinopathy, visual impairment and ocular status among patients with diabetes mellitus in Yemen: a hospital-based study. *Indian J Ophthalmol.* 2009;57:293–298. doi: 10.4103/0301-4738.53055.
3. Rani PK, Raman R, Chandrakantan A, Pal SS, Perumal GM, Sharma T. Risk factors for diabetic retinopathy in self-reported rural population with diabetes. *J Postgrad Med.* 2009;55:92–96. doi: 10.4103/0022-3859.48787.
4. Rodriguez-Fontal M, Kerrison JB, Alfaro DV, Jablon EP. Metabolic control and diabetic retinopathy. *Cur Diabetes Rev.* 2009;5:3–7. doi: 10.2174/157339909787314176.
5. Klein R, Klein BE, Moss SE, Davis MD, De Mets DL. The Wisconsin Epidemiologic Study of Diabetic Retinopathy. III. Prevalence and risk of diabetic retinopathy when age at diagnosis is 30 or more years. *Arch Ophthalmol.* 1984;102:527–32.
6. Klein R, Sharrett AR, Klein BE, et al. The association of atherosclerosis, vascular risk factors, and retinopathy in adults with diabetes: the Atherosclerosis Risk in Communities Study. *Ophthalmology.* 2002;109:1225–34.
7. Berinstein DM, Stahn RM, Welty TK, Leonardson GR, Herlihy JJ. The prevalence of diabetic retinopathy and associated risk factors among Sioux Indians. *Diabetes Care.* 1997;20:757–9.
8. UK Prospective Diabetes Study (UKPDS) Group. Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34) *Lancet.* 1998;352:854–865. doi: 10.1016/S0140-6736(98)07037-8.
9. Wang S, Xu L, Jonas JB, Wong TY, Cui T, Li Y, Wang YX, You QS, Yang H, Sun C. Major Eye Diseases and Risk Factors Associated with Systemic Hypertension in an Adult Chinese Population The Beijing Eye Study. *Ophthalmology.* 2009;116:2373–2380. doi: 10.1016/j.ophtha.2009.05.041.
10. Early photocoagulation for diabetic retinopathy. ETDRS report number 9. Early Treatment Diabetic Retinopathy Study Research Group. *Ophthalmology* 1991; 98 (5 Suppl):766S- 785
11. Cruickshanks KJ, Ritter LL, Klein R, Moss SE. The association of microalbuminuria with diabetic retinopathy: The Wisconsin Epidemiologic Study of Diabetic Retinopathy. *Ophthalmology.* 1993;100:862–7
12. Klein R, Moss SE, Klein BE. Is gross proteinuria a risk factor for the incidence of proliferative diabetic retinopathy? *Ophthalmology.* 1993;100:1140–6.

## (10) A STUDY ON MANAGEMENT OF EMERGENCY SERVICES AND CARE OF PATIENTS IN CORPORATE HOSPITAL OF AHMEDABAD

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### AIM AND OBJECTIVE:

- To provide the optimum level of emergency care in order to ensure maximum health and social gain for all patients to suffer accidental or sudden illness.

### OBJECTIVES

- To maintain quality of services provided to patients in emergency department.
- To evaluate the effectiveness of the care delivered in order to ensure optimal use of resources and identification of areas further improvement and development through clinical and service.
- To suggest measures to improve the hospital image for its clientele.

### METHODOLOGY

#### 1. Direct personal investigation

Direct method includes direct investigation with nursing staff, doctors and administrative staff of the emergency department for data collection to know the pattern of the working process.

#### 2. Questionnaire method

Used for the research study questionnaire copy is attached end of the project before the data analysis. Sample selection: Random sampling size of 50 patients.

#### 3. Secondary data

Collected from hospital brochures, registers, hospital records, internet and books from library.

### INTRODUCTION

The term "Emergency" is frequently used, particularly in modern hospitals. According to medical dictionary, emergency refers to unlooked for contingency or happening or a sudden demand for action or a situation requiring prompt action. "Casualty" as defined by Ministry of Health, London means a patient who comes to the hospital unannounced with accident injury and is seen and treated otherwise than at a consultative session.

Emergency Medical Care is progressively acquiring multifaceted dimensions. It has varied components like pre-hospital care, ambulance services, trauma care and it has to encounter medical and surgical emergencies of diverse forms. Emergency medicine, as a specialty is now beginning to develop in a number of countries throughout the world, and in fact it is a relatively new specialty even in countries where it is currently well established. Perhaps the best definition of the American College of Emergency Physicians is: "Emergency Medicine

encompasses the decision making and action necessary to prevent death or any further- disability for patients in health crises, as well as health promotion and injury prevention efforts Emergency medicine is an integral component of healthcare system.

Every Hospital, big or small, requires setting up a well organized emergency unit, because the image of hospital depends upon the quality and type of treatment a hospital can provide to a patient suffering from any medical or surgical complication requiring immediate care.

## **Overview of Emergency Department of Hospital**

It Is **LEVEL 1** participating hospital in the emergency Medical Services, Ahmedabad.

Equipped with the latest monitoring instruments, the department is all set to take care of any kind of emergency, round the clock. The department has **BLS (Basic Life Support)** and **ACLS (Advanced Cardiac Life Support)** trained doctors, nurses and technicians

### **Scope of Services**

- All kinds of medical and surgical emergencies
- Pediatric Emergencies & Obs & Gynec emergencies
- Polytrauma & Mass Casualty Management

### **Services Provided**

- Ambulance Services
- Organizing and Managing Camps
- PMS (Patient Movement Services) [Trolleys, Stretchers and Wheelchairs]

### **Location**

The emergency department of the Hospital is situated on ground floor with easy access to patients and ambulances. It has separate entrance, so patients coming in emergency do not face any delay by mixing with the general hospital population. It is well marked with proper lighting and signs. The functions of like admission, registrations of new patients are also done in the same department.

Hospital emergency department offers full time services in emergency care. The hospital's emergency department is open for 24 hours, 365 days in a year.

## **Physical facilities**

### **Inside Emergency room**

Total 10 beds in emergency room; which included 8 beds in emergency rooms with bed side multi para monitor, Oxygen flow meter and central suction line and two beds for observation in emergency room.

### **Outside Emergency room**

- Registration and admission office near the emergency room.
- Counseling room outside the emergency room for doctors.
- Space for stretchers and wheel chairs adjacent to entrance of emergency department.
- There are 6 stretchers and 6 wheel chairs outside the emergency room for shifting of indoor patients, patient from emergency room and outdoor patients to out patient

department, radiology, pathology or other department.

- Waiting room lounge for patient relatives with televisions, toilets, water coolers, STD, ISD, and PCO facilities, Photocopying machine, Tea and Coffee vending machines, bookstalls and lockers for patient relatives to keep their belongings.
- There is security office and ambulance office near emergency entrance gate.

### Emergency Department Questionnaire, Data Analysis and Findings:

- Alter arrival of your relative in the Emergency department, how long did you wait for doctor to assess your priority?
  - I did not have to wait to be assessed
  - 1-15 minutes
  - 16-30 minutes
- Did you have enough time to discuss your relative's health or medical problems /medical history with the doctor?
  - Yes, definitely
  - Yes to some extent
  - No
- While your relative were in emergency department, did the doctor kept your family and friends well informed about your relative's condition & treatment given to him and consent taken from you regarding any treatment related decision or reports to be carried out?
  - Yes, definitely
  - Yes to some extent
  - No
- After how many minutes - pain medicine is given to your relative?
  - 1-5 minutes
  - 5-15 minutes
  - 15-30 minutes
- How long did you wait for your tests (radiology and pathology investigation) to be carried out after arrival in emergency department?
  - 10-15 minutes
  - 15-30 minutes
  - More than 30 minutes
- Were you informed about the delay/reason of delay in carrying out radiology or pathology reports of your relative in emergency department?
  - Yes, definitely
  - Yes to some extent
  - No

- 7) Did the nursing staff/doctors inform you about the results of your relative's investigation in a way you could understand?
- (a) Yes, definitely
- (b) Yes to some extent
- (c) No
- 8) Did the nursing staff inform you about the medicine and injection given to your relative in emergency department?
- (a) Yes, definitely
- (b) Yes to some extent
- (c) No
- 9) Sometimes in hospital one staff member say one thing and another staff say something quite different; did this happen with you?
- (a) Yes, definitely
- (b) Yes to some extent
- (c) No
- 10) If your relative needs to be admitted how was over all attitude of the admission officer?
- (a) Good
- (b) Average
- (c) Poor
- 11) Did the "admission officer give proper information about room charges, facilities of the hospital and "other general information about the hospital?
- (a) Yes, definitely
- (b) Yes to some extent
- (c) No
- 12) Did the admission officer asked to pay initial deposit even in emergency?
- (a) Yes, definitely
- (b) Yes to some extent
- (c) No
- 13) Were MLC charges (Outside/Inside) explained by the admission officer: In case of medico legal cases?
- (a) Yes, definitely
- (b) Yes to some extent
- (c) No



- 14) After how much time police officer came to the hospital in case of medico legal cases?
- (a) Within 2 hr. of the arrival in the hospital
- (b) Within 6 hr. of the arrival in the hospital
- (c) Next day
- 15) If the patient is admitted after the short treatment in the emergency room. How was over all care taken by the emergency staff and doctor?
- (a) Good
- (b) Average
- (c) Poor
- 16) How long did it take to shift the patient after settle down in the room from the emergency department?
- (a) 15-20 minutes
- (b) 25-30 minutes
- (c) More than 1 hr.
- 17) In your opinion how clean was the emergency room?
- (a) Very clean
- (b) Fairly clean
- (c) Not very clean
- 18) Over all how would you rate the care you received in the Emergency department?
- (a) Excellent
- (b) Good
- (c) Average
- 19) Would you recommend any of your relative to visit this Hospital in case of any Emergency?
- (a) Yes, definitely
- (b) Yes to some extent
- (c) No

### Summary of Findings

- Almost all the patients coming to the emergency department got the services immediately and are given the immediate priority by the hospital staff.
- Most patients were given enough time to discuss their health problems with doctor/ nursing staff.
- Almost all relatives were kept well informed about the patient's condition and treatment given to him. Consent was also taken for all treatment related decisions. They were explained in simple manner about the nature of the treatment.
- Most of the patients got pain-killer medicine within 10 minutes after they ask for the medicine.

- Most of the patients got their tests done within 15 minutes of instruction given by the emergency registrar.
- Almost all relatives were informed about the delay in carrying out the test.
- Almost 30% relatives were found results of the tests explained in the way they understood, 60% relatives told the way is simple up to some extent and 10% relatives a way of explaining result was not proper and understandable.
- About 60% relatives were not given information about the medicines or injections given to their relatives.
- 75% of the patient relatives found that there was no contradiction between staff statements, 24% relatives found contradiction of statement between staff up to some extent and 1% found there was contradiction between staff statements.
- Almost all the relatives found attitude of the admission officer was good; admission officer has given proper information about facilities, rules, room charges and general facilities of the hospital.
- All the relatives were asked to pay initial deposit even in emergency.
- Medico Legal Charges were explained by the admission officer and consent was also taken for payment of the charges.
- Police arrived within 6 hour for verification in Medico Legal Cases.
- Almost all patients and relatives found the care and treatment given in the emergency department of the X Hospital was excellent.
- Most of the patients were shifted in rooms within 20-25 minutes.
- Almost all the relatives found the emergency department to be very clean and hygienic.
- All the relatives and patients would recommend visiting X Hospital for emergency treatment.

Overall, 80% patients & relatives found the emergency department's services to be excellent and 19% rated services as good. 1 % rated service as average, but all the patients and relatives assured that they would recommend people in need of emergency care to visit X Hospital for treatment

## **Recommendations**

Following are some recommendations to improve the quality of services in the emergency department and to achieve patient satisfaction.

### **For the staff of emergency department**

1. The hospital should give proper training to the ER registrars for the medico-legal cases
2. Whenever new nursing staff is recruited in the emergency department, he/she should be sent for specialized training in emergency services.
3. There should be a dress code for all the emergency staff as per their designation.
4. There should be a customer care executive for the emergency department to coordinate between patients, relatives, and nursing staff and ER registrar.
5. Nursing staff and Registrars of the Emergency department should be provided CME (Continuous Medical Education) to improve services and quality of the care.

## For the set-up of emergency department & ambulance

1. There should be a separate room (separate for men and women) for on duty doctors with sleeping accommodation and toilet facilities.
2. There should be an equipment room to put ventilators, suction machine, drugs and trolleys
3. There should be a separate room for the nursing staff with lockers facility.
4. There should be a provision for the expansion of the department, if required in future
5. There should be a suggestion box outside the emergency department for the Immediate feedback from patients and their relative that can help in improving the services levels effectively.
6. There should be proper communication system in the Ambulance.

## Conclusion

The study was conducted in the emergency department of Hospital to find out how the management with respect to the Emergency Department works in order to save the lives of the patients in need. The other Objectives are to maintain quality of services provided to patients in emergency department, to provide quick treatment to the patients and think upon implementing suggestive measures to improve the patient satisfaction and hospital image amongst its clientele.

At the end of the study it was concluded that emergency services of the hospital is good and quick. Staff and doctors are prompt in providing treatment to the patients who came in emergency and were successful in saving their lives. Patients were satisfied by the treatment given to them in emergency department. There are some gaps in the management; but that can be improved upon by following continuous staff training.

## (11) ASSOCIATION OF ABO BLOOD GROUPS WITH CORONARY ARTERY DISEASE IN GUJARATI POPULATION

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## Abstract

### Context:

Coronary artery disease (CAD) rates vary 10-fold among populations. The CAD rates among overseas Asian Indians worldwide are 50% to 400% higher than people of other ethnic origin irrespective of gender, religion, or social class. Due to inadequate information in the local population, the current study was conducted to find the association between ABO blood group system and coronary artery disease, so as to help in developing preventive strategies. A cross sectional study was conducted on 254 patients of coronary artery disease belonging to Gujarati community. Odds ratio was used to determine the relative risk of CAD association with ABO blood group system. A significant association was observed between CAD and ABO blood group system. Until now there is little study has undergone to find out association of ABO blood group with CAD (coronary artery disease) and different subgroup of patients of CAD having DM & HT, NON-DM & NON-HT, DM but NON-HT, HT but NON-DM.

### Aim:

To evaluate and find out association of ABO blood group on patients of coronary artery disease in Gujarati population.

### Result:

we observed that overall prevalence of ABO blood group in Local population was B blood group (33.93%), O (31.36%), A (24.39%) & AB (10.30%), while prevalence of ABO blood group in diseased people (people suffering from CAD) was O blood group (35.03%), B 30.70%), A (25.19%), AB (9.0%) different. Further, significant difference was noted between different subgroup of CAD.

### Conclusion:

Persons having blood group O and A have high relative risk of coronary artery disease as compared to persons having blood group B and AB. Early onset of CAD was seen in blood group A as compared to other blood groups.

**Keywords** – CAD - Coronary artery disease, CABG – coronary artery bypass grafting, CAG – coronary angiography, ABO blood group.

**TITLE: “Association of ABO blood group with coronary artery disease in Gujarati population”**

## Introduction

The ABO blood group system is widely credited to have been discovered by the Austrian scientist [Karl Landsteiner](#), who found three different blood types in 1900<sup>[1]</sup>. Blood group antigen represents genetic marker, which like other risk factors shows correlation with a number of diseases. In 1910, scientists proved that the RBCs antigens were inherited, and that the A and B antigens were inherited co dominantly over O<sup>[2]</sup>.

Alexander in 1921 showed higher incidence of various forms of cancer in blood group B and AB<sup>[3]</sup>. Aird et al (1953) correlated blood group A with stomach cancer<sup>[4]</sup>. Hembold (1961) also pointed towards the association of ABO blood group system and carcinoma of ovary<sup>[5]</sup>. As CAD

is of poly-etiological nature, ABO genes may play a significant predisposing factor in its causation.

A clear correlation has been established between the ABO phenotype and the level of two proteins involved in blood clotting; factor VII (FVIII) and von Willebrand factor (vWF) <sup>[6]</sup>. Blood group O individuals have about 25% less FVIII and vWF in their plasma. It is well established that low levels of FVIII and vWF are a cause of excess bleeding, and therefore it may also be the case that increased levels make clotting more likely, increasing the risk of both arterial (ischemic heart disease) and venous (thromboembolic disease) problems <sup>[7]</sup>. Indeed, non-group O individuals have been shown to be at an increased risk of both arterial and venous disease <sup>[8]</sup>.

### **Material and Methods:**

With this context present study was designed to find out association of ABO blood group in patients of coronary artery disease. An attempt is also made to find out prevalence of CAD in DM and HT patients. Study was conducted at cardiothoracic unit, in a reputed corporate hospital in western India. We included 254 patients in the study group diagnosed as CAD, who undergone CABG, following CAG, using strict inclusion and exclusion criteria.

### **Exclusion criteria:**

- Associated evidence of other disease apart from study subgroup. (e. g. patient of CAD having DM subgroup are not suffering from other disease apart from DM).

We randomly selected 660 people in the control group who are registered blood donors, free from disease in blood bank of government hospital to find out prevalence of blood group in local population.

All the patients selected for study are confirmed cases of CAD. Blood grouping were performed by standard method using antisera. An approval from institutional committee was obtained, Informed consent was taken from all the subjects and study was carried out in accordance with the world medical association declaration of Helsinki.

### **Statistical analysis of data**

Data was analyzed using SPSS version 13. Data was expressed as Mean, lower and upper limit and percentage. Chi-square test was applied to find out difference of statistical significance. P value < 0.1 was considered statistically significant.

### **Results**

Association of ABO blood group was determined against healthy volunteers free from disease, registered blood donors from the blood bank of government hospital in western India. Significant Association of ABO blood groups with coronary artery disease between different subgroup was seen.

254 subjects (Male + Female) with definite evidence of coronary artery disease with and without hypertension and/or diabetes mellitus with mean age and age range..... were analyzed.

**Table 1: Distribution of CAD cases according to Age and gender (n=254)**

Age group	Gender		Total	P value
	Male (%)	Female (%)		
31-40	09 (4.33)	00 (0.00)	09 (3.54)	0.01
41-50	25 (12.02)	01(2.17)	26( 10.23)	
51-60	90 (43.27)	17 (36.96)	107 (42.12)	
61-70	65 (31.25)	22 (47.83)	87 (34.12)	
71-80	19 (9.13)	06 (13.04)	25 (9.84)	
<b>Total</b>	208 (100.0)	46 (100.0)	254 (100.0)	

On applying Chi-square test, statistical significant association between age and sex of the CAD patients is found. (**p value= 0.01, df = 1**).

In younger age group (<50 years) males (n=32) suffers more than females (n=1) as age advances this ratio decreases. There were 133 (52.3%) cases in 4<sup>th</sup> and 5<sup>th</sup> decades and there was progressive increase no. of patients from 3<sup>rd</sup> decade onwards reaching maximum 107 (42%) in age group of 51–60 years. Thereafter decreasing trend was shown in successive later age groups as shown in table 1.

**Table 2: Distribution of CAD cases according to age and blood group (n=254)**

ABO Blood group	Age group					Total	P value
	31-40	41-50	51-60	61-70	71-80		
<b>A</b>	3	8	33	15	5	64 (25.20)	0.02
<b>B</b>	4	5	27	32	10	78 (30.70)	
<b>AB</b>	1	1	8	10	3	23 (9.06)	
<b>O</b>	1	12	39	30	7	89 (35.04)	
<b>Total</b>	9	26	107	87	25	254 (100.0)	

On applying Chi-square test, statistical significant association is found between age and blood group among the CAD patients. (**p value= 0.02, df = 3**). More than 50% cases in age group of 51 – 60 years in blood group A. more than 43% cases in age group of 51 – 60 years in blood group O.

**Table 3: Distribution of CAD cases according to gender and blood group (n=254)**

ABO Blood Group	Gender		Total	P value
	Male (%)	Female (%)		
<b>A</b>	54 (25.96)	10 (21.74)	64 (25.20)	0.7
<b>B</b>	65 (31.25)	13 (28.26)	78 (30.70)	
<b>AB</b>	17 (8.17)	6 (13.04)	23 (9.06)	
<b>O</b>	72 (34.62)	17 (36.96)	89 (35.04)	
<b>Total</b>	208 (100.0)	46 (100.0)	254 (100.0)	

On applying Chi-square test, it is seen that there is no statistical significant association between gender and blood group among the CAD patients. (**p value= 0.7, df = 3**).

Table 3 shows In A & B blood group males having higher percentages 25.96%, 31.25% respectively than females 21.74%, 28.26 of similar blood group out of total CAD patients, while for O & AB blood group it is reversed.

**Table 4: Distribution of ABO blood group among cases and control**

ABO Blood group	Study group	Control group
A	64 (25.20)	161 (24.39)
B	78 (30.70)	224 (33.93)
AB	23 (9.06)	68 (10.32)
O	89 (35.04)	207 (31.36)
<b>Total</b>	254 (100.0)	660 (100.0)

On applying Chi-square test, no statistical significant association is seen between cases and control in relation to blood group (**p value= 0.67, df= 3**).

Out of 660 controls selected 161 (24.39%) were of blood group A, 224 (33.93%) were of blood group B, 68 (10.30%) of blood group AB and 207 (31.36%) belonged to blood group O. In 254 cases 64 (25.14%) were in blood group A, 78 (30.70%) were in blood group B, 23 cases (9%) belonged to AB and 89 (35.03%) were in blood group O. so we see through strategically that although Blood group B is commonest in controls but in patients of coronary artery disease blood group O (35%) followed by blood group A (25%), blood group B was at 3<sup>rd</sup> place and AB was least common in cases. By strategic data there is high relative risk in blood group O (1.13) & blood group A (1.03) is seen.

**Table 5: ABO Blood group distribution among CAD cases in different subgroups**

ABO Blood group	CAD without DM and HT	CAD with DM and HT	CAD with DM only	CAD with HT only	Total
A	19 (29.68%)	25 (39.06%)	04 (6.25%)	16 (25%)	64 (100%)
B	22 (28.20%)	26 (33.33%)	16 (20.51%)	14 (17.94%)	78 (100%)
AB	05 (21.73%)	10 (43.47%)	03 (13.03%)	05 (21.73%)	23 (100%)
O	24 (26.96%)	26 (29.21%)	10 (11.23%)	29 (32.58%)	89 (100%)
<b>Total</b>	70	87	33	64	254

This study was conducted on 254 patients of confirmed case of coronary artery disease (CAD), among them 70 were NON-HT & NON-DM, 33 have DM (Diabetes Mellitus) but NON-HT, 64 having HT(Hypertension)but NON-DM, 87 having BOTH DM & HT, of confirmed case of coronary artery disease (CAD).

Table 5 shows patients having blood group A have higher percentages of patients those having no HT and DM, as compared to patients having only HT or only DM.

## Discussion

Coronary artery disease (CAD) (or [atherosclerotic heart disease](#)) is the end result of the accumulation of [atheromatous plaques](#) within the walls of the [coronary arteries](#) that supply the [myocardium](#) (the muscle of the [heart](#)) with oxygen and nutrients.

The prevalence of CAD has progressively increased in India during the latter half of the last century, particularly among the urban population<sup>[9]</sup>. The prevalence of CAD in urban India is about double<sup>[10]</sup> the rate in rural India, and about four fold higher than in the U.S.<sup>[11]</sup>

India is now in the middle of a CAD epidemic with urban Indians having CAD rates similar to overseas Indians, which is 4-fold higher than Americans. Whereas the CAD rates halved in the West in the past 30 years, the rates doubled in India with no signs of a downturn yet<sup>[12]</sup>.

The average age of first myocardial infarction (MI) has decreased by 20 years in India<sup>[13]</sup>. Among Asian Indian men, about half of all MI occur under the age of 50 and 25% under the age of 40.

This excess burden of premature CAD in Asian Indians is due to a genetic susceptibility, mediated through elevated levels of lipoprotein (a) {Lp (a)}<sup>[14]</sup>, which magnifies the adverse effects of lifestyle factors associated with urbanization, affluence, and changes in diet. Enas et al were the first to report high levels of Lp (a) in Asian Indians<sup>[15]</sup>. It appears that at a given level of any single or combination of conventional risk factor(s), the CAD rates among Asian Indians are at least double that of Whites. A more aggressive approach to prevention and treatment of both conventional and emerging risk factors is warranted in the Asian Indians. So, to find out genetic susceptibility in terms of ABO blood group and to begin preventive strategies at an earlier age is warranted because of extreme prematurity and malignant nature of CAD.

## Summary & Conclusions

In controls Blood group B was commonest followed by O and A while in CAD patients there was overall excess of blood group O and A (high relative risk is present).

Early onset of CAD was seen in blood group A and O compared to B and AB. Incidence of CAD rises upto the 6<sup>th</sup> decade were after falls in successive age groups, this may be due to poor survival.

In blood group A, those patients who are not suffering from HT & DM, have higher percentages than similar blood group of patient who is suffering from only HT or only DM.

Though there are multiple causation factors in Coronary Artery Disease, overall excess in blood group A, at very early age, warrants us for further studies and see for genetic aspects to find out the cause and early preventive strategies in cases of coronary artery disease.

## Bibliography

1. Landsteiner K. Weiner A. S. - Studies on an agglutininogen (Rh) in human blood reacting with antirhesus sera and with human isoantibodies. J. Exp. Med 74: 309,1941.



2. Crow J (1993). "Felix Bernstein and the first human marker locus". *Genetics* **133** (1): 4–7. [PMID8417988](#). Full text at [PMC: 1205297](#)
3. Alexander W. – An enquiry into the distribution of blood group in patients suffering from malignant diseases.
4. AIRD, J.; BENTALL, H. H.; and ROBERTS, J. A. F. 1953. A relationship between cancer of stomach and ABO blood groups. *Brit. Med. J.* 1: 794-801.
5. Hembold, Von W. – *Acta Genet (Basel)* 11:29, 1961.
6. Sarode R; Goldstein J, Sussman II, Nagel RL, Tsai HM (June 2000). "Role of A and B blood group antigens in the expression of adhesive activity of von Willebrand factor". *Br J Haematol.* **109** (4): 857–64.  
[doi:10.1046/j.1365-2141.2000.02113.x](#). [PMID10929042](#).
7. O'Donnell, J; Laffan MA (August 2001). "The relationship between ABO histo-blood group, factor VIII and von Willebrand factor". *Transfus Med.***11** (4): 343–51.  
[doi:10.1046/j.1365-3148.2001.00315.x](#). [PMID11532189](#).
8. *Blood*69 (6): 1691–5. [PMID3495304](#).  
<http://www.bloodjournal.org/cgi/content/abstract/69/6/1691>
9. Chaddha SL, Radhakrishnan S, Ramachandran K. Epidemiological study of coronary heart disease in urban population of New Delhi. *Ind J Med Res* 1990;92 (B):424-430.
10. Gupta R, Prakash H, Majumdar S, et al. Prevalence of coronary heart disease and coronary risk factors in urban population of Rajasthan, India. *Indian Heart J* 1995; 47:331-338.
11. Decline in deaths from heart disease and stroke-United States, 1900-1999. *MMWR* 1999;48:649-656.
12. Reddy KS. Rising burden of cardiovascular diseases in India. In: Sethi KK, ed. *Coronary Artery Disease in Indians: A Global Perspective*. Mumbai: Cardiology Society of India, 1998:63-72.
13. Enas EA. Avoiding premature coronary deaths in British Asians: Guidelines for pharmacologic intervention are needed. *BMJ* 1996;312:376.
14. Enas EA, Yusuf S, Garg A, Davidson L, Thomas J, Pearson T. Lipoprotein (a) levels in Indian physicians: Comparison with Black and White physicians in the U.S.A. *Indian Heart J* 1994;46 suppl:185(abstract).
15. Enas EA. Lipoprotein (a) is an important genetic risk factor for coronary artery disease in Asian Indians. *Am J Cardiol* 2001;88:201-202.

## **(12) STUDY OF PULMONARY FUNCTIONS IN AIR CONDITIONER USERS AND NON USERS**

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### **INTRODUCTION :**

Air conditioning may affect the human health since it has profound effect on our environment than just lowering temperature. Hyperventilation of cold dry air causes bronchoconstriction in asthmatic patients. Cold dry air is what we inhale while using AC's; hence alteration in pulmonary functions may also be simulated in AC users. Modern style of living in urban area is responsible for airway allergic diseases.

### **OBJECTIVE :**

This study was planned to observe the effect of air condition (AC) on pulmonary functions.

### **MATERIALS AND METHODS :**

The study group contain 30 subjects who were using AC in their cars for at least 2 hours daily since last 6 months and 30 subjects who did not use AC as a controls. All the subjects are male and age group between 20 to 30 years. The pulmonary functions were assessed using spirometer in room.

Experimental protocol was explained and consent was taken. History was taken and clinical examination (general and systemic) done for inclusion and exclusion criteria. Statistical analysis was done by unpaired t test. P value less than 0.05 considered as a significant.

### **RESULT :**

Age, Height and Weight are not statistically significant in study group and controls. Forced vital capacity (FVC) and Forced expiratory volume in 1 second (FEV<sub>1</sub>) are not significant in both group. Peak expiratory flow rate (PEFR), Forced expiratory flow (FEF) are significantly decreased in AC users. FEF<sub>25-75%</sub> which is the flow rate over the middle half of the forced vital capacity (FVC) is significantly decreased in AC users.

### **CONCLUSION :**

Significant decrease in PEFR and FEF<sub>25-75%</sub> in AC users predispose towards the mild respiratory limitation.

**KEY WORDS :** Air conditioner, PEFR, FEF<sub>25-75%</sub>  
Pulmonary functions, FEV<sub>1</sub>

## **MAIN ARTICLE**

### **INTRODUCTION :**

One of the component in modern lifestyle is intensive use of AC's, which has increased the risk of atopic sensitization. Air conditioners (AC) are used extensively these days indoor as well as while travelling. The air inside is cooled at the expense of air outside. The reduction in humidity

of the air being cooled is due to the condensation of water vapours. It has been observed that hyperventilation of cold dry air causes bronchoconstriction in asthmatic patients. Modern styles of living in urban areas have been considered potentially responsible for the development of airway allergic diseases due to proliferating house dust mites & increasing concentration of indoor air pollutants, which lead to the elevation of serum IgE levels or the enhancement of eosinophil activity. (1, 2)

Increased prevalence of IgG induced sensitization and hypersensitivity pneumonitis is reported in persons exposed to aerosols of contaminated AC's. While fluorinated hydrocarbons collectively referred as Freon have been shown to result in widespread toxicity after accidental or intentional inhalation. Freon inhalations may lead to the production of cardiac arrhythmias. (3) Freon primarily serve as propellants and are widely used in cooling systems. Therefore we planned to evaluate the lung function tests of young healthy non-smokers using car AC's.

### **MATERIALS AND METHODS :**

The study group contain 30 subjects who were using AC in their cars for at least 2 hours daily since last 6 months and 30 subjects who did not use AC as a control. All the subjects are male and age group between 20 to 30 years. The pulmonary functions were assessed using spirometer in room.

Experimental protocol was explained and consent was taken. History was taken and clinical examination (general and systemic) done for inclusion and exclusion criteria.

### **INCLUSION CRITERIA :**

- (1) Male with age group of 20-30 years.
- (2) Healthy individual without any known disease.
- (3) No history of drug, smoking or any condition that affect pulmonary functions.

### **EXCLUSION CRITERIA :**

- (1) Individual with any known general or systemic disease.
- (2) Any history of drug or addiction that affect the pulmonary functions.
- (3) Using AC on irregular basis.
- (4) Any un co-operative subject.

### **EXPERIMENTAL PROTOCOL :**

- (1) All the subjects filled the consent form that they are willing to participate in the study.
- (2) History and clinical examination was done for inclusion and exclusion criteria.
- (3) Pulmonary function test was done by spirometer in room with sitting posture and using nose clip.
- (4) Then mean value and standard deviation was estimated by statistical method.

### **PROCEDURE :**

- (1) Pulmonary function test was done by spirometer with inbuilt computer program.
- (2) The subject was given proper instruction by us for procedure.
- (3) Subject was seated in comfortable seating position.
- (4) Mouthpiece and nose clip applied to subject during procedure.

**STATISTICAL ANALYSIS :**

Unpaired t test is used for analysis and P value less than 0.05 consider as a significant.

**RESULT :**

Data presented are Mean  $\pm$  SD (standard deviation)

**Table 1: Anthropometric parameters**

PARAMETERS	AC USERS	NON AC USERS	SIGNIFICANCE
Age (years)	24.32 $\pm$ 1.63	24.18 $\pm$ 1.64	NS
Height (cms)	164.68 $\pm$ 9.42	165.22 $\pm$ 8.63	NS
Weight (kg)	62.72 $\pm$ 5.95	62.84 $\pm$ 6.77	NS

S: Significant (P value less than 0.05)

NS: Not significant (P value more than 0.05)

**Table 2: Lung volumes and capacities (liters)**

PARAMETERS	AC USERS	NON AC USERS	SIGNIFICANCE
FEV <sub>1</sub>	3.34 $\pm$ 0.74	4.09 $\pm$ 1.13	NS
FVC	3.79 $\pm$ 0.86	4.22 $\pm$ 1.26	NS
FEV <sub>1</sub> /FVC	90.41 $\pm$ 15.87	97.46 $\pm$ 0.93	NS

**Table 3: Pulmonary flow rates (liters/min)**

PARAMETERS	AC USERS	NON AC USERS	SIGNIFICANCE
PEFR	6.18 $\pm$ 2.30	9.05 $\pm$ 3.57	S
FEF <sub>25%</sub>	5.66 $\pm$ 2.48	8.51 $\pm$ 2.97	S
FEF <sub>75%</sub>	2.92 $\pm$ 1.08	3.92 $\pm$ 0.82	S
FEF <sub>25-75%</sub>	4.38 $\pm$ 1.66	6.05 $\pm$ 1.27	S

Age, Height and Weight are not statistically significant in study group and controls. Forced vital capacity (FVC) and Forced expiratory volume in 1 second (FEV<sub>1</sub>) are not significant in both group. FEV<sub>1</sub>/FVC ratio is also not significant. Peak expiratory flow rate (PEFR), Forced expiratory flow (FEF) are significantly decreased in AC users. FEF<sub>25-75%</sub> which is the flow rate over the middle half of the forced vital capacity (FVC) is significantly decreased in AC users.

**DISCUSSION**

The results of the present study show a predisposition of AC users towards respiratory dysfunction. (9) There is a definite impairment in the expiratory flow rates especially the FEF<sub>25-75%</sub>, which is the flow rate over the middle half of the forced vital capacity (FVC), In the presence of normal FEV<sub>1</sub>, reduced FEF<sub>25-75%</sub> is an evidence for mild airflow limitation. This index is recommended as a screening test for mild airflow limitation.

PEFR reflects mainly the caliber of the bronchi and larger bronchioles, which are subjected to

reflex bronchoconstriction. Bronchoconstriction in asthmatic subjects has been reported on hyperventilation of cold dry air. (3, 4, 5)

All the subjects of the present study were driving their cars themselves, the direction of the blow of air was directly on their face, which may be one of the reasons for these changes. (6) Repeated cooling and dessication of peripheral airways can cause airway remodeling similar to that seen in asthma. Crude water extracts of contaminated AC's are the antigen-source of the hypersensitivity pneumonitis. (7, 8)

Our subjects used AC's in their cars during the hot humid environment, which is the climate prone for the growth of various allergens. Further studies are require on large scales basis for confirmation of our study.

## CONCLUSION

Significant decrease in PEFR and FEF25-75% in AC users predispose towards the mild respiratory limitation.

## REFERENCES

- (1) Caire N, Cartier A and Malo GL. Influence of the duration of cold dry air on the resulting bronchoconstriction in asthmatic subjects. *Eur Respir J* 1989 ; 2: 741-745.
- (2) Carpentar GB, Win GR, Massey DJ. Air conditioning and house dust mite. *J Allergy Clin Immunol* 1985; 75 : 121.
- (3) Desjardins A, De Luca S, Cartier A. Non specific bronchial hyper responsive to inhaled histamine. *Am Rev Respir Dis* 1988 ; 137: 1020-1025.
- (4) Hosein HR, Corey P, Robertson J Mc D. The effect of domestic factors on respiratory symptoms and FEV1. *Int J Epidemiology* 1989; 18(2): 390–396.
- (5) Kumar P, Marier R, Leech SH. Hypersensitivity pneumonitis due to contamination of an air conditioner. *N Engl J Med* 1970; 283: 271-276.
- (6) Scharf SM, Heimer D, Walters M. Bronchial challenge with room temperature isocapnic hyperventilation. *Chest* 1985; 88: 586-593.
- (7) Takaoka M. House dust mites in the Japanese indoor environment. *Allergology* 1997; 4: 367–373.
- (8) Van Strien RT, Gehring U, Belanger K, Triche E, Gent J, Bracken MB, Leaderer BP. The influence of air conditioning, humidity, temperature and other household characteristics on mite allergen concentrations in the northeastern United States. *Allergy* 2004 June; 59(6): 645–652.
- (9) Welty C, Weiss ST, Tager IB. The relationship of airway responsiveness to cold air, cigarette smoking and atopy to respiratory symptoms and pulmonary function in adults. *Am Rev Respire Dis* 1984; 130: 198-203.

## (13) CORNEOSCLERAL PATCH GRAFT IN A CASE OF MOOREN'S ULCER : A CASE REPORT

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### **Abstract**

Mooren's ulcer is a rare disorder of unknown etiology that is refractory to treatment. It can affect not just the cornea but also the scleral tissue and can involve both eyes. We report a case of a 25-year-old young male with a history of bilateral and malignant Mooren's ulcer. The patient had lost left eye to perforation of Mooren's ulcer before 3 years. The perforated Mooren's corneal ulcer also presented in the right eye and involved the adjacent scleral tissue. A crescent shaped corneal-scleral patch graft was performed to preserve the anatomical integrity of the eye as well as vision along with local as well as systemic immunosuppression. This report highlights how a corneal-scleral patch graft followed by systemic and local immunosuppressive treatment can be considered in monocular patients with malignant Mooren's ulcer which can prove to be globe saving as well as vision saving.

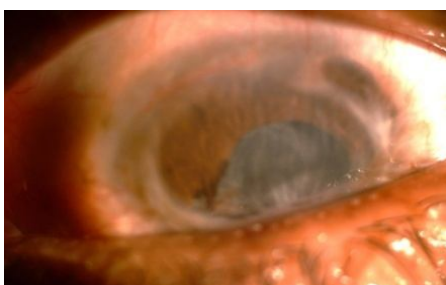
**Key words :** Mooren's ulcer, patch graft

### **Introduction**

Mooren's ulcer is a rare disorder presenting as chronic and painful ulceration of the cornea. The lesion with overhanging edges generally starts on the periphery and tends to spread progressively to the entire circumference or towards the centre of the cornea. As well as the cornea, the sclera can also be involved with an incidence of 13.5% of eye perforation and loss of vision [1]. We describe a case of crescent shaped patch grafting in a patient with Mooren's ulcer and corneoscleral perforation inferiorly.

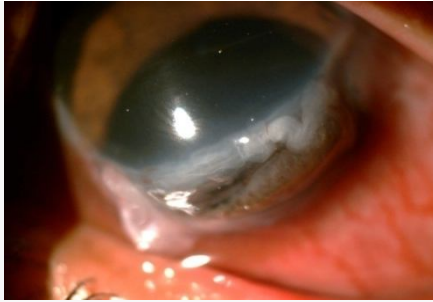
### **Case presentation**

A 25-year-old male presented at our clinic with a 2 day history of loss of vision in his right eye associated with redness, watering, pain and photophobia since 1 week. 3 years back he had an episode of similar symptoms in the left eye following which he lost left eye which had then become pthisical.



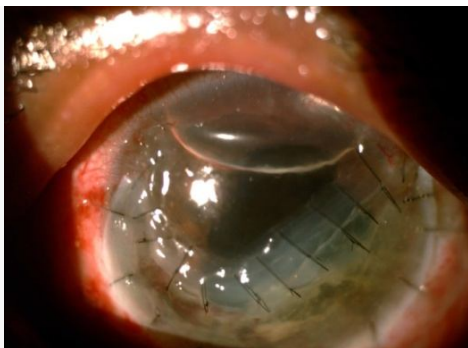
**Figure 1 : Pthisical left eye**

Medical examination of right eye revealed acute conjunctival hyperaemia and a large inferiorly perforated Mooren's ulcer extending almost from 3 to 8 o'clock hours. The anterior chamber was shallow with iris prolapse. Patient was aphakic with orange glow (probably patient had trivial trauma superimposed on the existing condition leading to a large perforation with lens extrusion) (Figure 1). Visual acuity was hand movements.

**Figure 2. Perforated Mooren's ulcer.**

Under general anaesthesia, we performed an inferior crescent shaped corneoscleral patch graft. The surgical technique included conjunctival peritomy inferiorly to explore extent of perforation and involvement of sclera. The anterior chamber was formed with viscoelastics and prolapsed iris was repositioned. The involved cornea and scleral ring was removed. The recipient bed was measured and donor tissue prepared from a whole eye used within 24 hours of the donor's death and stored in a moist chamber using free hand dissection. Care was taken to scrape off the uveal tissue from the donor tissue. The donor corneoscleral graft was then sutured into place onto the scleral edge using 8.0 interrupted silk sutures and corneal edge using 10.0 interrupted ethilon sutures. The anterior chamber was washed with balanced salt solution (BSS) and filled with air. Patient was kept aphakic. The conjunctiva was closed with Vicryl 8.0 interrupted sutures.

The day after surgery we found a mild keratitis with a low flare in the anterior chamber. Postoperative steroid therapy was prednisolone acetate 1% every two hours for four weeks (decreased to four times a day for five months), oral cyclosporine A (5 mg/kg/day) for six months and prednisone (started at 2 mg/kg/day tapered gradually over 12 months). After six months the best corrected visual acuity was 6/60.

**Figure 3. Corneoscleral patch graft after 3 days.**

## Conclusion and Discussion

The presented case was a malignant form of bilateral Mooren's ulcer that had already caused a corneal perforation in the left eye with consequent phthisis bulbi. Patient presented with similar perforation in the right eye and so a corneoscleral graft was necessary. The etiopathogenesis of Mooren's ulcer is still unknown. It is probably an autoimmune disease. Indeed, anti-corneal and anti-conjunctival tissue antibodies have been isolated from patients suffering from this disorder [2]. Furthermore, the conjunctival tissue surrounding the lesion is generally rich in proteo-glycolytic enzymes secreted by mononucleate cells and neutrophils that progressively infiltrate the area surrounding the ulcer [3]. Another confirmation of the autoimmune origin of the disorder is the T-suppressor lymphocytes deficit in patient blood samples [4].

Wood and Kaufman classified Mooren's ulcer in two main forms [5]. Type 1 is the benign form, generally monolateral, which mainly affects the elderly. The symptoms are unclear but this type responds well to medical and surgical management. Type 2 is the malignant form. It can occur bilaterally in 25% of cases in white subjects and 75% in black subjects. It mainly affects younger individuals. Watson divided the disease into three types based on the clinical presentation: unilateral Mooren's ulcer, bilateral aggressive Mooren's ulcer and bilateral indolent Mooren's ulcer [6].

In the third type of Watson and in the malignant form of Wood and Kaufmann, we have a progressive ulcer that affects not just the cornea but also the scleral tissue. For this reason, neither conventional medical nor surgical treatment is sufficient, and it can be necessary to perform a large corneoscleral graft to preserve the anatomical integrity of the eye.

Indeed, it is extremely difficult to treat Mooren's ulcer and in many cases, the results are poor. Treatment starts with cortisone administered systemically and locally, but if this is unsuccessful, the complete excision of the perilimbal conjunctiva and episclera near the ulcer is made [6]. The use of immunosuppressive drugs, in particular cyclosporin A, should be reserved for more severe forms [7]. In the event of small corneal perforation, the area can be covered with amniotic membrane or a lamellar keratoplasty can be performed [8]. In this case, because of the presence of a large, perforated corneal ulcer affecting the scleral tissue and the patient being one eyed, it was decided to perform a corneoscleral patch graft.

The use of total corneoscleral grafts to treat serious corneal disorders was first proposed in the early 1970s [9]. The percentage of surviving graft tissue has always been very low mainly because of the early onset of epithelial damage to the transplanted tissue, recurrence of the underlying disorder or secondary glaucoma caused by the disturbance of the iridocorneal angle and thereby of aqueous humour filtering [10]. In this case, the particular technique used made it possible to preserve the iridocorneal angle, and the administration of systemic and local cyclosporin treatment avoided the onset of secondary glaucoma as well as any sign of rejection six months after the operation. For this reason, we think that this type of graft can be proposed mainly for monocular patients with malignant Mooren's ulcer where there is high risk and where eyes would otherwise be condemned to a complete loss of vision.



## References

1. Jiaqi Chen, Hanping Xie, Zhen Wang, Bing Yang, Zuguo Liu, Longshan Chen, Xiangming Gong, Yuesheng Lin: **Mooren's ulcer in China: a study of clinical characteristics and treatment.** Br J Ophthalmol 2000, **84**: 1244-49. [Publisher full text](#)
2. Schaap OL, Feltkamp TEW, Breehart AC: **Circulating antibodies to corneal tissue in a patient suffering from Mooren's ulcer (ulcers rodens cornea).** Clin Exp Immunol. 1969, **5**: 365-72.
3. Brown SL: **Mooren's ulcer: histopathology and proteolytic enzymes of adjacent conjunctiva.** Br J Ophthalmol 1975, **59**:670-4. [Publisher Full Text](#)
4. Murray PI, Rahi AHS: **Pathogenesis of Mooren's ulcer: some new concepts.** Br J Ophthalmol 1984, **68**:182-7. [Publisher Full Text](#)
5. Wood TO, Kaufman HE: **Mooren's ulcer.** Am J Ophthalmol 1971, **71**:417-22.
6. Watson PG: **Management of Mooren's ulceration.** Eye 1997, **11**:349-56.
7. Hemady R, Tauber J, Foster CS: **Immunosuppressive drugs in immune and inflammatory ocular disease.** Surv Ophthalmol 1991, **35**:369-85. [Publisher Full Text](#)
8. Nian ZD, et al.: **Mooren's ulcer treated by lamellar keratoplasty.** Jpn J Ophthalmol 1979, **15**:290-4.
9. Ticho U, Ben-Sira I: **Total keratoplasty.** Arch Ophthalmol 1973, **90**:104-6.
10. Cobo M, Ortiz JR, Safran SG: **Sclerokeratoplasty with maintenance of the angle.** Am J Ophthalmol 1992, **113**:533-7.

## (14) EFFECT OF DEEP BREATHING ON HEART RATE VARIABILITY IN NORMOTENSIVE MALE OFFSPRING'S OF HYPERTENSIVE PARENTS

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## ABSTRACT

### Background :

Hypertension runs in families, and parental history of hypertension increases the risk of developing hypertension, especially if both the parents are hypertensive's. In all, 25% of children with one hypertensive parent and 50% of children with two hypertensive parents will eventually become hypertensive demonstrating that heredity plays a major role in the development of the disease. The present study was undertaken to test the hypothesis that normotensive young male adults, with parental history of hypertension exhibit variations in cardiovascular autonomic functions as tested by the analysis of HRV with deep breathing.

### Material And Methods :

The study group of 25 healthy normotensive male subjects whose parents are hypertensive, either father or mother or both being hypertensive but none being diabetic whereas control group of 25 healthy normotensive male subjects whose parents were normotensive and non diabetic. All are aged between 18 to 25 years.

### Results :

There was no significant difference in the anthropometric measurements and the resting parameters between the two groups ( $p>0.05$ ). The Heart Rate Variability with Deep Breathing (HRVdb) was reduced in the study group when compared to the control group and was statistically significant ( $p<0.04$ ).

### Conclusions :

The study shows that HRV is reduced in normotensive young men with parenteral history of hypertension. Even though the baseline cardiovascular parameters values were normal in both the groups, it was by the way of recording of HRV with rest and the autonomic challenge by the way of the HRVdb the autonomic imbalance could be made out in the study group.

**Key Words :** HRV db, Family history of Hypertension, Heart Rate, Blood pressure

## INTRODUCTION

Hypertension is the most common human cardiovascular disease. Worldwide it is estimated to cause 7.1 million premature deaths each year and 4.5% of the disease burden<sup>1</sup>.

Hypertension runs in families, and parental history of hypertension increases the risk of developing hypertension, especially if both the parents are hypertensive's<sup>2</sup>. In all, 25% of children with one hypertensive parent and 50% of children with two hypertensive parents will eventually become hypertensive demonstrating that heredity plays a major role in the development of the disease<sup>3</sup>.

The ANS plays a fundamental role in the control of arterial blood pressure (BP) and, therefore, may be considered an important pathophysiologic factor in the development of arterial hypertension<sup>4</sup>. Normotensive subjects with family history of hypertension have greater sympathetic activity and also early parasympathetic attenuation<sup>5</sup>.

HRV has emerged as a practical, noninvasive tool to quantitatively assess cardiac autonomic dysfunction in hypertension<sup>6</sup>. Research into HRV and respiration over the past 150 years has

led to the insight that HRV with deep breathing (HRVdb) is a highly sensitive measure of parasympathetic cardiac function. HRVdb is a reliable clinical test for early detection of cardiovascular dysfunction in a wide range of autonomic disorders<sup>7</sup>.

The present study was undertaken to test the hypothesis that normotensive young male adults, with parental history of hypertension exhibit variations in cardiovascular autonomic functions as tested by the analysis of HRV with deep breathing as compared to age and sex matched adults without family history of hypertension.

## **MATERIAL AND METHODS :**

The present study was conducted in the Research laboratory in the Department of Physiology, Navodaya Medical College, Raichur, Karnataka.

The study group consisted of 25 healthy normotensive male subjects studying medical students (aged between 18 to 25 years) whose parents are hypertensive, either father or mother or both being hypertensive but none being diabetic.

The control group consisted of 25 healthy normotensive male subjects studying medical students (aged between 18 to 25 years) whose parents are normotensive and non diabetic.

The study was approved by Ethical Committee, Navodaya Medical College, Raichur.

**Inclusion Criteria :** (common to both study and control groups)

- Healthy males in the age group of 18–25 yrs, non smokers, non alcoholic and normal range of BMI between 18.5 – 24.9 kg/m<sup>2</sup>
- Parental hypertension > 10 yrs

**Exclusion Criteria :** H/O any systemic disorders known to cause to autonomic dysfunction.

## **Study protocol**

The protocol was explained to the subjects and informed consent was obtained from each of the participant.

The participants were called between 10 to 12 AM after 2 hours of light breakfast. They were advised to avoid consumption of tea\coffee 12 hours prior to reporting in the laboratory. Upon arrival the subjects were made to sit in the lab comfortably for 10 minutes to get accustomed to the new environment. Height in meters and weight in kgs were measured and BMI calculated. Then they were allowed to rest supine for fifteen minutes. The baseline RR, HR, SBP and DBP were recorded.

## **Heart Rate Variability Analysis**

Recording were standardized and instructions followed as per the guidelines of Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology.

A chest lead ECG was recorded using ECG V: 52 [HRV analysis software] for 5 minutes in supine rest with eyes closed, which is simultaneously analyzed by the software.

Beat-to-beat variations in instantaneous HR were derived offline using a rate-detector algorithm. For computing HRV indices during supine rest, recommendations of the Task Force on HRV were followed. Briefly, a 5-min ECG was acquired at a sampling rate of 1000 Hz

during supine rest with the subjects breathing normally at 12–18 per min. RR intervals were plotted using the ECG V: 52software. An RR series was extracted using a rate-detector algorithm after exclusion of artifacts and ectopic. A stationary 256 second RR series was chosen for analysis. In the time domain, the standard deviation of normal-to-normal RR intervals (SDNN) was taken as an index of overall HRV. Low frequency (LF) and high frequency (HF) spectral powers were determined by integrating the power spectrum between 0.04 and 0.15 Hz and 0.15 and 0.4 Hz respectively. Total power was calculated by integrating the spectrum between 0.004 and 0.4 Hz and includes very low frequency, LF and HF components. Spectral powers are expressed in absolute units of milliseconds squared. LF and HF powers are also expressed in normalized units.

### Heart Rate Response to Deep Breathing:

The subject is trained to breathe deeply at a rate of 6 breaths /minute in supine position. The subject is asked to breathe deeply, steadily and slowly for 1min at the rate of 6 breathes /min (5 sec inspiration and 5 sec expiration duration of each cycle of one minute) while ECG was continuously recorded. The HR change with deep breathing (deep breathing difference) was then expressed as the mean of the differences between the maximal and minimal HR in 6 respiratory cycles. Deep Breathing Difference (DBD) = Mean of HR Differences in 6 Breath Cycles.

### Statistical analysis:

All data is expressed as Mean  $\pm$  SD. Student 't' test used to compare the data of both the groups. Mann-Whitney test used to analyze the Heart Rate Variability (HRV). The association between HRV and the variables (Age, BMI, Rest HR, SBP, and DBP) analyzed by stepwise multiple regressions. A p-value  $<$  0.05 considered statistically significant and p value  $<$  0.01 as statistically highly significant.

**Results:** There was no significant difference in the anthropometric measurements and the resting parameters between the two groups. The results are depicted in Table 1.

**Table-1:** Baseline Characteristics of Subjects (Mean  $\pm$  SD)

Parameters	Study group	Control group	t-value	p-value	Remarks
Age (years)	19.65 $\pm$ 1.63	18.90 $\pm$ 1.80	1.38	0.18	Not significant
BMI (Kg/m <sup>2</sup> )	21.85 $\pm$ 2.93	21.00 $\pm$ 1.64	1.13	0.27	Not significant
Resting RR breath/min	13.72 $\pm$ 0.6	14.02 $\pm$ 0.7	1.36	0.18	Not significant
Resting HR (bpm)	71.58 $\pm$ 5.48	75.66 $\pm$ 11.65	1.42	0.16	Not significant
Resting SBP (mm Hg)	121.70 $\pm$ 2.99	121.40 $\pm$ 6.8	0.18	0.86	Not significant
Resting DBP (mm Hg)	79.10 $\pm$ 2.63	77.30 $\pm$ 4.55	1.53	0.13	Not significant

**Note:** BMI - Body mass index, RR - Respiratory rate, HR - Heart rate, SBP - Systolic blood pressure, DBP - Diastolic blood pressure.

### Heart Rate Variability (HRV) Parameters

The measured values of HRV parameters like Mean RRI, RMSSD, LF, LFnu, HF, HF nu and LF/HF among both the groups are presented in the Table-2. LFnu power indicating sympathetic activity were increased in the study group. HF & HFnu power indicating parasympathetic activity were decreased in study group. The LF/HF ratio indicating statistically sympathovagal balance was increased in study group. But the difference was not significant which exhibit little alteration in cardiovascular autonomic function.

**Table-2:** HRV Parameters (Mean  $\pm$  SD) in Study group and Control group.

Parameter	Study group	Control group	U-value	p value	Remarks
MEAN RRI	767.45 $\pm$ 53.88	811.70 $\pm$ 115.61	147.5	0.13	Not significant
RMSSD	23.66 $\pm$ 5.02	27.25 $\pm$ 9.96	173.5	0.16	Not significant
LF	908.95 $\pm$ 122.07	986.85 $\pm$ 168.62	133.5	0.10	Not significant
LF nu	68.10 $\pm$ 3.7	65.26 $\pm$ 7.07	152.5	0.12	Not significant
HF	445.48 $\pm$ 46.35	474.68 $\pm$ 58.48	147.0	0.09	Not significant
HF nu	31.9 $\pm$ 1.90	33.08 $\pm$ 1.97	128.55	0.07	Not significant
LF/HF	2.06 $\pm$ 0.38	2.10 $\pm$ 0.42	183.5	0.08	Not significant

**RMSSD** : Root mean square successive difference in milliseconds, **LF** : Low frequency, **LF nu** : Low frequency in normalized units, **HF** High Frequency, **HF nu** : High Frequency in normalized units, **LF/HF** : Low frequency/ High Frequency ratio.

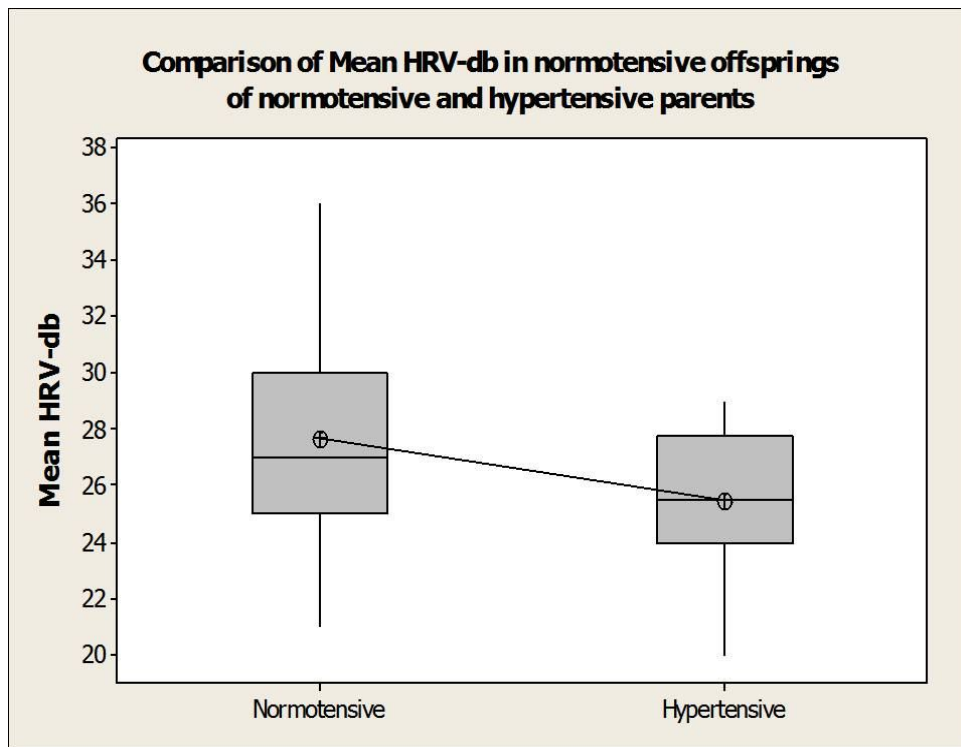
### HRV with Deep Breathing:

The measured value of Inspiration-Expiration (I-E) HR difference with deep breathing in Study group and Control group subjects, expressed as Mean  $\pm$  SD is presented in the Table-3.

**Table-3:** HRVdb (Mean  $\pm$  SD) in Study group and Control group

Parameter	Study group	Control group	U-value	p value	Remarks
HRVdb	25.05 $\pm$ 2.46	27.70 $\pm$ 3.98	113.0	0.04	Significant

The Heart Rate Variability with Deep Breathing (HRVdb) was reduced in the study group when compared to the control group and was statistically significant ( $p < 0.04$ ).



### Discussion :

Both groups in this study consisted of age, sex, and BMI-matched, young, normotensive male subjects. They differed only in the genetic propensity for developing hypertension later in life. Although the resting heart rate, systolic and diastolic blood pressures were found to be higher in subjects of study group as compared with control group this difference was statistically insignificant ( $p > 0.05$ ) (Table I). This shows that both the groups are normotensive at rest.

Similar baseline characteristics were observed in studies done by Chinagudi. S et al<sup>9</sup> and Rathi. P et al<sup>10</sup>. This may be due to the younger age group and the BMI was within normal limits.

However studies done by Lopes et al<sup>11</sup> and Ostfeld et al<sup>12</sup> report a slightly elevated pressure blood in normotensive subjects with a family history of parental hypertension. This blood pressure elevation may be considered as a permanent abnormality characterizing a prehypertensive stage early in life<sup>13</sup>. Study done by Julis et al opines that those with parental history of hypertension their offspring show higher resting diastolic pressure probably due to hyperactive sympathetic nervous system (SNS)<sup>14</sup>.

### Measures of Heart Rate Variability (HRV):

The time domain methods are used to investigate recordings of short durations, whereas the frequency domain methods are usually able to provide results that are more easily interpretable in terms of physiological regulations.

In our study the measures namely the time domain measures of HRV, the Mean RRI and RMSSD were reduced in the study group when compared to the control group. The Mean RRI measures the sum of the levels of parasympathetic and sympathetic influences and RMSSD reflects an estimate of parasympathetic regulation of the heart<sup>15</sup>.

Also among the frequency domain measures of HRV our study showed us that there is LFnu and decreased HFnu and increased LF/HF ratio in the study group when compared with controls. Similar results have been reported by other investigators<sup>9,16,17</sup>. LFnu reflects the sympathetic activity while HFnu is the direct representation of vagal tone and LF/HF ratio is an indicator of sympathovagal imbalance<sup>15</sup>. These findings indicate that there is increased sympathetic activity and decreased parasympathetic activity in the study group when compared with the control group.

### **Heart Rate Variability with Deep Breathing (HRVdb):**

In our study the Heart Rate Variability with Deep Breathing (HRVdb) was reduced in the study group when compared to the control group and was statistically significant. Similar result was observed in the study done by Krishnan et al<sup>17</sup>

Deep breathing induced changes in HR occur because of alterations in cardiac parasympathetic activity and when this system is impaired deep breathing leads to decrease in HRV.

### **Three mechanisms are generally proposed to explain the modulation of HR associated with respiration :**

1. A direct influence of medullary respiratory neurons on cardio motor neurons;
2. An indirect influence on heart rate of blood pressure changes secondary to respiratory movements that are mediated via arterial baroreceptors or atrial stretch receptors;
3. A reflex response to lung inflation mediated by thoracic stretch receptors, most likely from the lungs and chest wall.

In most autonomic disorders, parasympathetic function is affected before sympathetic function, so HRVdb provides a sensitive screening measure for parasympathetic dysfunction in many autonomic disorders<sup>18</sup>.

### **Conclusion:**

In conclusion, this study shows that HRV is reduced in normotensive young men with parenteral history of hypertension. Even though the baseline cardiovascular parameters values were normal in both the groups, it was by the way of recording of HRV with rest and the autonomic challenge by the way of the HRVdb the autonomic imbalance could be made out in the study group. Since the subjects are young, the autonomic imbalance in them suggests that tendency for developing hypertension sets in at an early age. So in such individuals the regular monitoring of autonomic activity by simple noninvasive recording of HRV and by simple parasympathetic test like HRVdb may prove to be a useful tool in predicting the future hypertensive. Also as a preventive measure, the predisposed group should aim at decreasing the sympathetic drive and increasing their parasympathetic drive which can be achieved by lifestyle modifications and slow breathing exercises<sup>12,13</sup>.

### **REFERENCES :**

1. World Health Organization (2003) and International Society of Hypertension statement on management of hypertension. World Health Organization and International Society of Hypertension writing, group. Journal of Hypertension. November 2003; 21(11):1983-1992.

2. Wang NY, Young JH, Meoni LA, Ford DE, Erlinger TP, Klag MJ. Blood pressure change and risk of hypertension associated with parental hypertension: the john's Hopkins precursor study. *Archives of Internal Medicine* 2008; 168(6): 643-648.
3. Schneider GM, Jacobs DW, Gevirtz RN and O'Connor DT. Cardiovascular hemodynamic response to repeated mental stress in normotensive subjects at genetic risk of hypertension: evidence of enhanced reactivity, blunted adaptation, and delayed recovery. *Journal of human hypertension* 2003; 17: 829-840.
4. Davidson's Principals and Practice of medicine, 20th edition, Hypertension, part 2,609.
5. Stolraz K, Grodzicki T, Lubaszawski W, et al. Heart rate variability in offspring of hypertensive parents. *Przegl Lek.* 2002; 59: 892-94.
6. Emily B. Schroeder, Duanping Liao, Lloyd E. Chambless, Ronald J. Prineas, Gregory W. Evans, Gerardo Heiss. Hypertension, Blood Pressure, and Heart Rate Variability; The Atherosclerosis Risk in Communities (ARIC) Study. *Hypertension.* 2003; 42: 1106-1111.
7. Robert W Shields. Heart rate variability with deep breathing as a clinical test of cardiovagal function. *Cleveland Clinical Journal of Medicine.* April 2009; 76:S37-S40.
8. Surekharani Chinagudi, Anita Herur, Shailaja Patil, Shashikala G V, Roopa A; Comparative study of Heart Rate Variability in normotensive offsprings of hypertensive parents. *Biomedical Research;* 24 (1): 123-126.
9. Preeti Rathi, Vinay Agarwal and Ajay Kumar; Sympathetic hyperactivity in children of hypertensive parents; *ANNALS OF NEUROSCIENCES VOLUME 20 NUMBER 1 JANUARY 2013.*
10. H. F. Lopes et al; Autonomic abnormalities demonstrable in young normotensive subjects who are children of hypertensive parents; *Brazilian Journal of Medical and Biological Research* (2000) 33: 51-54.
11. Ostfeld AM & Oglesby P (1963). The inheritance of hypertension. *Lancet*, 1: 575-579.
12. Ravogli A, Trazzi S, Villani A, Mutti E, Cuspidi C, Sampieri L, De Ambroggi L, Parati G, Zanchetti A & Mancia G (1990). Early 24-hour blood pressure elevation in normotensive subjects with parental hypertension. *Hypertension*, 16: 491-497.
13. Steve Julius, Autonomic Nervous System Dysregulation in Human Hypertension; *Am J Cardiol* 1661; 67:36-7B).
14. Task Force Report: 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. *Circulation* 1990; 93: 1043-1065.
15. Pal GK, Pal P, Nanda N, Lalita V, Datta TK, Adithan C. Sympathovagal Imbalance in Prehypertensive. Offsprings of Two Parents versus One Parent Hypertensive. *International Journal of Hypertension* 2011; doi:10.4061/2011/263170.



16. Krishnan Muralikrishnan, Kabali Balasubramanian and Badanidiyur Vishwanatha Rao. Heart Rate Variability In Normotensive Subjects With Family History of Hypertension. Indian J Physiol Pharmacol 2011; 55(3):253-261.
17. Judith Ann Hirsch And Beverly Bishop, Respiratory sinus arrhythmia in humans: how breathing pattern modulates heart rate, American journal of Physiol. 241. 1981; H620-H629.
18. Pramanik T, Sharma HO, Mishra S, Mishra A, Prajapathi R and Singh S. Immediate Effect of Slow Pace Bhastrika Pranayama on Blood Pressure and Heart Rate. Journal of Alternative and Complimentary Medicine 2009; 15(3): 293-295.

## **(15) IMPACT OF GENDER/SEX ON ACADEMIC EXAMINATION RELATED STRESS SYMPTOM PROFILE IN SPECIAL REFERENCE TO SLEEP AMONG MEDICAL STUDENTS.**

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### **ABSTRACT :**

#### **OBJECTIVE :**

This study was done to examine the prevalence of psycho- physiological parameters and pattern of parameters among male and female students due to examination stress and to study the impact of gender/sex on academic examination related stress symptom profile in special reference to sleep and insomnia among medical students during one medical academic examination of first year student.

#### **MATERIALS AND METHODS :**

This class room based study was carried out on one hundred and fifteen first-year students [88 male and 27 female]. A self-administered questionnaire and one established Stress Scale to assess and stress related neuro behavioral Physiological parameters like sleep, appetite, ability to concentration, decision making... was given to students to before one academic examination and the prevalence of symptoms of stress were assessed and compared among both male female students in special reference to sleep problems.

**RESULT :**

There were 115 positive responses out of 150 students suggesting significant stress >The positive stress among students was 76% i.e. 76% of study group have significant stress related to examination. The most common symptoms were problems related to sleep i.e. Insomnia, bodily pains, poor concentration, decision making, worrying among both male and females group but sleep and pain were significant high among female group. Abnormal sleep habits, with a statistically significant increase in female students ( $p =$ ). Their Abnormal sleep pattern defined as subjective feeling of insufficient sleep duration, delayed sleep onset, and occurrence of napping episodes during the day.

**CONCLUSION :**

76% of students were documents significant stress during examination period. A high prevalence of abnormal sleep pattern i.e. Insomnia was found among both male and female group of students and significant more among female students. Because of examination related stress and insomnia some students prone to benzodiazepine group of drugs and later on may become dependent on such drugs. Instead of such unhealthy coping strategies, healthy coping strategies /skills are recommended for coping such problems i.e. relaxation, meditation, yoga, pranayam, light music and some spiritual prayers.

**KEY WORDS :** Examination stress, abnormal sleep pattern, medical students

**Introduction:**

Examination stress is a feeling of pressure tension that many young medical students feel before and coming up to academic examination time. It usually occurs during the revision period before examinations and immediately before and during the examinations themselves. Many students perceive stress during such academic examination time. And the first MBBS medical students are considered as a unique group due to first exploration in medical science, peer pressure and competition, expectations from staff and parents, change in environment, change in languages, uncertainty of oral and practical examinations, limitation of students to cope and so many other factors. Though moderate amount of stress is essential for maintaining motto for better performance and preparation in examination, extreme stress can be harmful to body and mind. Usually stress is defines as nonspecific response to unusual excessive demand. The responses depend upon how student perceive as excessive demand and how he/she response as a unique organism. Stress is often associated with occurrence of various neuro behaviors symptoms related autonomic, cardiovascular and central nervous system. We as a faculty members have witnessed extreme responses to such stress from severe depression, avoidance of examination i.e. drop out to the extreme rare incidences of suicides during carrier of as medical UG and PG students. The majority of students cope well to examination stress with some symptoms which are fairly manageable with much intervention i.e. counseling. Our college department is running a counseling and Yoga centre for managing such problems. This study was done to evaluate prevalence of stress among 1st year medical students, the impact of examination stress on psycho- physiological parameters and pattern of parameters among male and female students and to study the impact of gender/sex on academic examination related stress symptom profile in special reference to sleep/insomnia

among medical students during one medical academic examination of first year student. Past studies have found that many of such symptoms i.e. problem of sleep, concentration... are associated with poor academic performance. So it is highly recommended that such symptoms of stress are not neglected, perceived by counseling and managed well by ideal methods i.e. proper counseling and yoga- meditation ect.. and by unhealthy methods i.e. by drugs and tobacco and other bad habits avoided by students.

### **MATERIALS AND METHODS :**

This classroom based study comprises of 150 1st year MBBS students of mean age 19 +- 0.8 years. The protocol was explained during an academic lecture on Stress physiology and management just before preliminary examination. At the end of the lecture the stress assessment scale questionnaire was given to fill voluntarily to all students irrespective of gender /sex without any compulsion. Score more than 20 [Table 1] were considered as stressful state while score less than 20 were stress free and have adequate coping strategies..

**Table 1: Scale used to study the stress**

Symptom	Very frequently	Frequently	Sometimes	Rarely	Never
Sleep problems Insomnia	4	3	2	1	0
Pains, e.g., headache, back					
Extreme variations in appetite					
Reduced self-confidence					
Difficulty in making decisions					
Problems concentrating					
Worrying more than usual					
Increased irritability					
Feeling miserable					
Withdrawing from contact					

### **Scoring**

Allocating scores of 4 (very frequently) to 0 (never), it is possible to loosely quantify experiences of physiological stress. Scores of 30-40 indicate high levels and may be attributable to ongoing circumstances in which the individual might benefit from seeking support. Scores of 20-29 indicate moderate strain which the individual should monitor to avoid escalation of these levels. Scores below 20 are likelier to be within individual's coping capabilities.

Insomnia was defined by the presence of an individual's report of difficulty with sleep. (1) difficulty falling asleep, staying asleep or no restorative sleep; (2) this difficulty is present despite adequate opportunity and circumstance to sleep; (3) this impairment in sleep is

associated with daytime impairment or distress; and (4) this sleep difficulty occurs at least 3 times per week and has been a problem for at least 1 month.

The pattern of symptoms of stress were assessed separately and compared among male and female students Table 2:

### Results:

There were 115 out of 150 have a responses consistent with positive for stress with percentage rate of 76%. Among them 115 were male and 27 females showing positive for stress scale.

**Table 2: Symptoms of stress in number and % were assessed separately and compared among male and female students**

		Females students			Male students			P	Sig. y/n
		Present	%	N	Present	%	N		
1	sleeplessness/Insomnia	25	92.5	27	56	63.84	88	0.0034	Y
2	Pains, e.g., headache, back	21	77.7	27	16	18.24	88	0.0001	Y
3	variations in appetite	15	55.5	27	17	19.38	88	0.0005	Y
4	Reduced self-confidence	18	66.6	27	27	30.78	88	0.0014	Y
5	Difficulty in making decisions	12	44.4	27	45	51.3	88	0.2783	NS
6	Problems in concentrating	22	81.4	27	50	56.8	88	0.0235	Y
7	Worrying more than usual	14	51.8	27	48	54.12	88	0.8286	NS
8	Increased irritability	16	59.2	27	41	46.74	88	0.2779	NS
9	Feeling miserable	11	40.7	27	2	2.28	88	0.0001	Y
10	Withdrawing from contact	1	3.7	27	1	0	88	0.4160	Y

From table, it is evident that insomnia and pains was commonest symptoms among both groups of students and were significantly more among female students.

### Discussion:

76% of students experience significant stress before examinations. Why examination is a stressful event is highly an individual perception of students. There is exceptional students who take examination as a challenge or fun. But majority of students to think the examination is stressful event as it is evident it is necessary to work hard to remember and recall a large amount of information for an examination. Even and above there always uncertainty of result of examination with fear of parental and peer Pressure due to competition. Certain symptoms are very specific during examination stress i.e. feeling low, feeling butterflies in your stomach, having a racing heart beat or feeling sick, feeling confused, losing touch with friends and the hobbies or even going mind blank during the test.

The most important symptoms of examination stress in this study was poor sleep up to the extent of insomnia. In the study 92.5 % of female students and 63.84 % of male students have documented insomnia. There many studies stated that poor sleep is associated with poor performance in examination. At Tel Aviv University, Dr. Avi Sadeh conducted a study of students. He found that those "who tended to focus on their emotions and anxiety during the high-stress period were more likely to shorten their sleep, while those who tended to ignore emotions and focus on tasks extended their sleep and shut themselves off from stress." "This reason why female have more sleep problems compared to male students as female brains are considered as more often emotion focused rather than task. Sleep quality improved or remained the same for students who directed their focus away from their emotions." "If you can't cope with it, sleep on it," Sadeh said "

Sleep problems may, in turn, contribute to other psycho physiological problems. Insomnia may increase an individual's risk of developing depression or anxiety. The emotional and psychological responses of medical students at Stanford University School of Medicine. A large fraction of the students reported sleep problems. In 2005 NSF *Sleep in America* poll of all adults revealed that women are more likely than men to have difficulty falling and staying asleep and to experience more daytime sleepiness at least a few nights/days a week. Research has shown. This is because the changing levels of hormones that a woman experiences throughout the month and over her lifetime, like estrogen and progesterone, have an impact on sleep. Understanding the effects of these hormones, environmental factors and lifestyle habits can help enjoy a good night's sleep and more studies are in this field.

*It is worth to ention that Sleep Deprivation in students leads to **excessive fatigue and tiredness, Clumsiness, Tardiness or absenteeism at college, bodily pain; general health immune problems i.e. cold and viral fevers; lack of vitality; poor social functioning; anxiety and difficulty in concentration. Last point i.e. concentration and health** is very crucial for medical students for preparing medical examination. And this creates the stress-poor sleep-stress. Vicious cycle i.e.*

At molecular level it is found that sleep disturbance are related to over activation of the hypothalamic-pituitary-adrenal (HPA) axis and the hypersecretion of cortisol. This further agravete the stress response. Hypothalamic-pituitary-adrenal (HPA) axis and cortisol are centre pillar of stress response. Cortisol i.e. glucocorticoids suppress the secretion of two molecules i.e. Interleukin-1 and tumor necrotic factor TNF. These molecules i.e. Interleukin-1 and tumor necrotic factor TNF are considered to be the last cytokine signals to initial and maintain sleep at neuronal level and suppression of these molecules lead to insomnia. Patients taking glucocorticoid group of drugs have documented insomnias in many studies. In this context it is possible comment that in future eg, corticotrophin releasing hormone [CRH] antagonists may be ideal therapeutic to manage both stress and insomnia. The neuroendocrine system during stress and insomnia provide evidence of arousal as demonstrated by chronic activation of the stress response system. Several studies measuring 24-hour urinary free cortisol excretion have found high levels among insomnias. Urinary free cortisol levels have also been positively correlated with total wake time. Plasma measures of cortisol and adrenocorricotropic hormone (ACTH) have been evaluated in poor and healthy normal sleepers. Poor sleepers have higher levels of these compounds in their plasma. Positron emission tomography (PET) has been used to assess cerebral glucose metabolism, an indirect measure of whole brain metabolism, in

insomnia. Compared to healthy subjects, during insomnia greater cerebral glucose metabolism was observed during waking and non-rapid eye movement (REM) sleep states.

### Conclusion:

76% of student was documented significant stress during examination period. A high prevalence of abnormal sleep pattern i.e. Insomnia was found among both male and female group of students and significant more among female students. More studies are required to find the answer why females students are more prone to insomnia. Sleep problems i.e. insomnia occur in medical students in conjunction with many other environmental/behavioral causes, including: Poor habits, Poor diet, Lack of guidance, Assertion of independence, Anxiety, Changes associated with moving or home life. Some recommendations to students in this context are Sleep hygiene, regular bedtime hours, avoiding of caffeinated beverages, eliminating television, video games and computer use and distraction at sleep time, healthy diet and exercise, Keep the bedroom dark at bedtime, Avoid napping too much. As poor sleep is associated with poor academic performance in many studies. It is highly recommended to students to go for healthy coping strategies /skills for coping i.e. relaxation, meditation, yoga, pranayam, light music and some spiritual practices. Because of examination related stress and insomnia some time medical students prone to go for unhealthy coping straggles i.e. benzodiazepine group of drugs and later on may become dependent on such drugs. Such consequences are prevented by counseling and guidance in time. It is well said catch them in time before it is too late.

### References :

1. **Abdulghani HM**, Alrowais NA, Bin-Saad NS, Al-Subaie NM, Haji AM, Alhaqwi AI. Sleep disorder among medical students: relationship to their academic performance. Med Teach. 2012;34 Suppl 1:S37-41. doi: 10.3109/0142159X.2012.656749. PMID: 22409189
2. Bonnet MH, Arand DL. Heart rate variability in insomniacs and matched normal sleepers. Psychosom Med. 1998;60:610–5. [PubMed] Vgontzas AN, Tsigos C, Bixler EO, Stratakis CA, Zachman K, Kales A, Vela-Bueno A, Chrousos GP. Chronic insomnia and activity of the stress system: a preliminary study. J Psychosom Res. 1998;45:21–31. [PubMed]
3. **Christopher J. Davis, PhD, James M. Krueger, PhD\***, **Sleep and Cytokines A Book : Biology of sleep-sleep medicine clinics. Sept. 2012, vol.7 - no. 3, published by Elsevier publication**
4. Kuppermann M, Lubeck DP, Mazonson PD, Patrick DL, Stewart AL, Buesching DP, Fifer SK. Sleep problems and their correlates in a working population. J Gen Intern Med. 1995;10:25–32. [PubMed]
5. Michael Perlis, Wil Pigeon, Phil Gehrman, Jim Findley, Sean Drummond, **Neurobiological mechanisms in chronic insomnia, A Book: Biology of sleep-sleep medicine clinics. Sept. 2012, vol.7 - no. 3, published by Elsevier publication**
6. Neckelmann, D. et al., Chronic Insomnia as a Risk Factor for Developing Anxiety and Depression, Sleep. 2007; 30 (7): 873-880. Ibid.
7. Roberts R, Roberts CR, Chan W. Persistence and Change in Symptoms of Insomnia among Adolescents. SLEEP 2008;31(2): 177-184.

## (16) ANTEVERSION OF ADULT FEMORA IN GUJARAT POPULATION

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### **ABSTRACT**

#### **Introduction :**

Among all the bones femur is most attended to. The most conspicuous peculiarity of femur being anteversion of neck which goes hand in hand with the evolution of the erect posture. Femoral anteversion (torsion) is the lateral rotation of the neck of femur to the long axis of its shaft. The objective measurement of this angle is of paramount importance in total hip replacement surgery for attaining the normal activity and longevity of the replaced joint. It is an important factor for the performance and longevity of these joints too. Angle of anteversion is measured in adult Gujarat dried femora. The present study is an attempt to evaluate the normal anteversion range in adult Gujarat femora.

#### **Material :**

Two hundred ossified femora belonging to skeletons of unknown age, sex, and stature from Gujarat population were used in this study. 200 femora were studied from anatomy department of Smt. N.H.L municipal medical college, Ahmedabad. Out of 200 femora, 123 were of right side and 77 were of left side.

#### **Method :**

The angle of femoral anteversion was measured by a method described by Pearson and Bell (1919). The axis of the knee was taken by placing the femur with the posterior surfaces of the condyles and the trochanter touching the surface of the osteometric board. In this position the posterior surfaces of the femoral condyles and the plane of the horizontal surface of the board give serviceable approximation to the orientation of the transverse axis of the knee as both are parallel.

#### **Observations :**

The angle of anteversion in the present series was 9.7 degrees which was quite lower figure than English figures (15 degrees). Range of variation in anteversion angle was from 31 to -16 which is very wide. There was no significant difference in mean values of angle of torsion on either side in the entire series.

#### **Discussion :**

On comparison with the series of other workers there was variation in the average means of the series. It revealed that maximum value of mean was 30.6 degree (Fair bank 1930) and minimum as 8.10 degrees (Kingsley and Olmsted 1984). Kate and Robert (1963) have examined 104 bones and the mean average calculated was 8.8 degrees while that of our series of 200

femora was 9.7 degree. The range of variation was 51 degree to -12 degree. According to LeDamany (1903 & 1914) and Altmann (1924) in foetal life the angle of femoral anteversion is negative i.e. at 17 mm stage – 10 degree and at 60 mm stage 0 degree and just after 4<sup>th</sup> month 12 degree. The percentage of retroversion calculated was 7.50 in the present study and was nearly the same as reported by Kate and Robert (1963). On comparison with others it revealed that it was less in the series of Lofgren, while frequent and significant in the series of Kingsley and Olmsted (1948).

### **Conclusions :**

Clinical studies would possibly show some similarity or difference of angle on two sides. The mean angle for left (9.09 degrees) has been recorded lesser than right side (10.08 degrees). The angle of retroversion was present in 7.50%.

### **Introduction :**

Orthopedists and anatomists have long found interest in the anteversion of femur. Both groups have made deep studies on dried macerated femora as well as on living subjects. The knowledge of the angle of femoral anteversion guides the treatment of congenital dislocation of hip, perthe's disease and malunited fractures of the femur. The abnormal range has been attributed as a direct cause of this disability.

The gray's anatomy states that the transverse axis of the head of femur makes an angle of approximately 15 degrees with the transverse condylar axis and orthopedics books quote it to range from 10 to 30 degrees. Thus, literature reveals variable values of femoral anteversion without mention about the impact of origin of the population on these values.

### **Material and Method :**

Present study was conducted with aim to measure the angle of femoral anteversion on 200 femora.

The mechanical axis of the neck was taken as the line joining the two points namely the centre of the head where thickness is maximum and the centre of the neck where antero-posterior thickness is least when viewed from above. A pin was stuck with plastic in along the line of these two points (fig1). The femur was then placed against the vertical wall of osteometric board with a paper pinned to wall.

Corresponding to the pin the neck axis line was drawn on the paper, while the lower edge of the paper remained parallel to the surface of the board which corresponds with the axis of the knee. The angle thus formed between lower edge of the paper and the pin line gives the angle of femoral anteversion.

When the angle is negative the plotting of the axis of the neck along the pin instead of remaining above the base line which represents the axis of the lower end goes below it in the reverse fashion (fig2).

After measuring the angle in all femora the average mean, range and standard deviation were calculated and side wise variations and incidence of retroversion noted. The data were statistically analyzed and compared with series of other workers to draw the conclusions.





**Fig 1:** Showing pin stuck with plastic in along the long axis of the neck.



**Fig 2:** Showing standard position of the femur for plotting of neck axis on the pinned paper (Retroversion).

### Observations:

**Table 1** Showing the angle of femoral anteversion of total 200 bones.

Femora	200
Mean	9.7 degrees
S.E.M	$\pm 0.60$
Range of variation	31 to -16
S.D.	8.50

Table one shows, the average mean angle of femoral anteversion for the total of 200 femora was found to be 9.7 degrees which is lower than the usually thought of 12 degrees and standard error of mean (S.E.M.)  $\pm 0.60$  and standard deviation (S.D.) 8.50 degrees. The range of variations of the femoral anteversion was from 31 to -16 degrees.

**Table 2** Showing difference on right and left side.

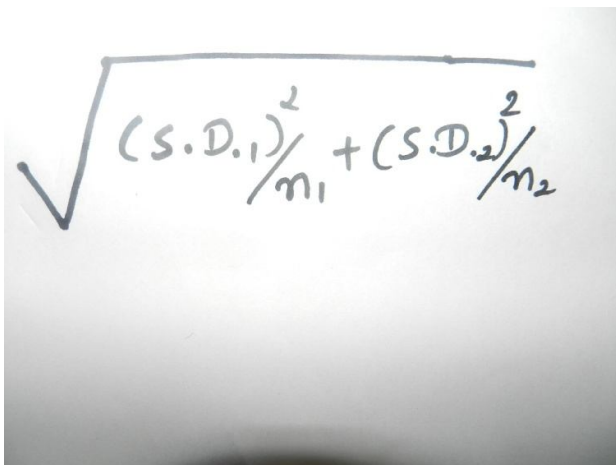
	Right	left
Femora	123	77
Mean	10.08 degrees	9.09 degrees
S.E.M.	0.748	0.981
Range of variation	31 to -16 degrees	31 to -16 degrees
S.D.	8.4	8.61

Table 2 shows average mean of 123 right side femora was 10.08 degree, S.E.M.  $\pm$  0.838 and S.D. was 8.4 degree. The mean of 77 left sided femora was 9.09 degree, S.E.M.  $\pm$  0.981 and S.D. was 8.61 degrees. The angle on left side is seen to be lesser than that on right side. The range of variations for right and left femora was from 31 to -16 degrees.

**Table 3** Showing large sample test to assess the significance of the difference in the means of right and left side.

Side	Mean in degree	Difference in mean	S.E. difference	Interference
Left	Femora 77 Mean 9.09 S.D. 8.6	0.99	1.53	Not significant
Right	Femora 123 Mean 10.08 S.D. 8.4			

S.E. difference between two means:



$$\sqrt{(S.D.1)^2/n_1 + (S.D.2)^2/n_2}$$

Where S.D.1 = Standard deviation of right side femora  
 S.D.2 = Standard deviation of left side femora  
 n1 = no. of bones of right femora  
 n2 = no. of bones of left femora

Sample test was found to be not significant because two means not differ by more than twice the value of S.E. difference.

## Discussion:

On comparison with the series of other workers there was variation in the average means of the series. It revealed that maximum value of mean was 30.6 degree (Fair bank 1930) and minimum as 8.10 degrees (Kingsley and Olmsted 1984).

It has been observed that the variation from the means and large number of bones go hand in hand. Kate and Robert (1963) have examined 104 bones and the mean average calculated was 8.8 degrees while that of my series of 200 femora was 9.7 degree. The range of variation was 51 degree to -12 degree. Average mean of present series was more towards that of Kate and Robert partly because of same method used. It is also not very convincing in the study of Kate and Robert (1963) that their mean is quite lower i.e. 8.8 degree and is almost the same as that of Kingsley and Olmsted (1948), while the methods used by these two different authors are entirely different. Hence to give the lower of femoral anteversion a racial character or to associate it with squatting habits also does not stand to reason in view of our study where mean is as high as reported by Pearson and Bell (1919).

According to Le Damany (1903 & 1914) and Altmann (1924) in foetal life the angle of femoral anteversion is negative i.e. at 17 mm stage – 10 degree and at 60 mm stage 0 degree and just after 4<sup>th</sup> month 12 degree. They stated that during development, neck of femur rotates with respect to condyles. They further stated that this progressive changes occur till birth, which are attributed to the disposition of the foetus in the uterine cavity. Here, acute flexion of the hip takes place, and with further difficulty in the adjustment the neck of the femur rotates on the shaft, (these force results in internal rotation of the shaft) resulting in anteversion.

It is well established fact that detorsion takes place in the first two years of life when child starts walking and with weight bearing till the adult figure of 12 degree is reached. After birth extrinsic factors like pull of external rotators, action of gluteus maximus, capsular restriction, support of the weight of the body in walking and weight of the lower extremity itself during first year partially rectify anteversion to adult stage.

Increased incidence of a greater angle of femoral anteversion may be present in squatters, as due to squatting there may be cessation in the decrease in femoral anteversion. There is some similarity in squatting position and intrauterine position of the foetus. The squatting may interfere with the extrinsic factors and reduced the decrease in the degree of femoral anteversion.

Figure for total 200 femora revealed that 108 fall in the range of 5 to 15 degree and 45 bones in the range of 15 and 25 degree. It revealed that frequency distribution is very wide. The high degree of anteversion more than 10 degree is infatible type (more towards pathological, Kingsley and Olmsted 1948).

It is interesting to note that with lower degree of anteversion the incidence of congenital dislocation of hip may be less common in Indians but so far no clinical data is available to support this hypothesis. If this factor plays any role than the angle of femoral anteversion should be much more than 12 degree or more than that observed by western workers on whites but in this study the angle is 9.7. This fact shows that squatting habit may not in any way affect the angle of femoral anteversion.

In the present study range was from 31 to -16 degree. On comparison with others it revealed that in all the series the range is wide and wide range is associated with larger number of bones. The difference between the means of present study and the series of others were S.D. was available has been evaluated by applying large sample test. It revealed significance only in the case of Pearson and Bell (1919) though the method used was the same, which may be due to the influence of the environmental habits, nutritional and endocrinal factors. In present study mean angle on left side was less than that of right. The difference between the left and right may be attributed to organic asymmetry (Pearson and Bell 1919). This fact is also confirmed by applying large sample test which is found not significant. Thus, the observed difference is due to mere chance.

Greater right side anteversion has been documented by Le Damany (1903), Kingsley et al (1948) and Yoshioka et al (1987) UN Caucasians, Oriental and African population. This bilateral limb asymmetry should discourage the tendency to view the lower limb as mirror images of one another.

The percentage of retroversion calculated was 7.50 in the present study and was nearly the same as reported by Kate and Robert (1963) and Ingalls (1948). On comparison with others it revealed that it was less in the series of Lofgren, while frequent and significant in the series of Kingsley and Olmsted (1948) did not give any reason about the frequent and significant incidence of retroversion in their series. But Lofgren (1956) suggested that this might be due to high living standard, sedentary habits and more uses of motorcars by Americans.

Kate and Robert (1963) stated that frequent occurrence of retroversion might be due to arrest of development of the angle towards the primitive form of original i.e. -10 degree at 17 mm stage, or the process of detorsion occurring during first 2 years carried on further unchecked due to developmental, mechanical and endocrinal factors, the nutritional factor like scurvy and ricket might be more responsible than others. Parson (1914) suggested that it is exaggerated due to twisting of lower end outwards instead of inwards. Moss and Noback (1958) stated that mechanical and acquired postural habits are possibly responsible for retroversion.

### **Conclusions:**

The angle of femoral anteversion in the present series was 9.7 degree which was quite lower figure than the English figures (15 degree). There was no significant difference in mean values of angle of femoral anteversion on either side in the entire series. However, the question whether there is any difference of anteversion on two sides in each individual person cannot be answered from such study.

Clinical studies would possibly show some similarity or difference of angle on two sides.

### **References:**

1. Altman, F(1924). Untersuchungen uber die Torsio Femories. Zeit. Anat, Entwick; 75, 82-126. Cited by Elftman (1945).
2. Fairbank (1930), Congenital dislocation of hips with special reference to anatomy. Br. J. Surg. 17, 380-416.

3. Gray's anatomy 38<sup>th</sup> edition (1996).
4. Kate B.R. and Robert, S.L. (1963), The angle of femoral torsion. J. Anat. Soc. of India, 12, 8-11.
5. Kingsley and Olmsted (1948), A study to determine the angle of anteversion of neck of femur. J. bone Jt. Surg. 30 A, 745-751.
6. Le Damany (1903): les torsions osseuses leur role dans la transformation des membres. Journal of Anatomical Physiology. 39: 426-450.
7. Moss and Noback (1958), Cited by Roberts, Walter (1962).
8. Pearson, K and Bell J. (1919) A study of long bones of the English skeleton Drapers Col research Memoirs, biometric series 10 ad 11 par 1(text).
9. Parson, F.G. (1914) The characters of the English thigh bones. J. Anat. Physiol. Lond., 48, 238-267.
10. Yoshioka, Y, Siu, D. and Cooke, T.D.V. (1987): The anatomy of functional axis of the femur. Journal of Bone and Joint Surgery. 69 : 873-880.

## **(17) EVALUATION OF ABDOMINAL SACROPEXY FOR VAGINA VAULT PROLAPSE**

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**ABSTRACT :**

**OBJECTIVE (S) :** To evaluate the Abdominal Sacropexy for the correction of vaginal vault prolapse.

**METHOD (S) :** Ten patients of vaginal vault prolapse were subjected to Abdominal Sacropexy. The post operative depth of vagina, axis of vagina and other post operative complications related to surgery were observed.

**RESULT (S) :** The Abdominal approach for vaginal vault prolapse in form of Abdominal Sacropexy was successful with adequate vaginal depth and proper vaginal axis without any post operative complains.

**CONCLUSION (S) :** Abdominal Sacropexy offers reliable fixation of the vaginal apex with minimal changes to the vaginal anatomy.

**KEY WORDS :** abdominal sacropexy, vaginal vault prolapse, vaginal axis

**INTRODUCTION :**

As women live longer and healthier lives, pelvis disorders continue to become even more prevalent. The ultimate goal of pelvic reconstructive surgery is to restore anatomy, maintain or restore visceral and normal sexual functions. The main goal of vault prolapse repair aims to suspend the vaginal vault as near as possible to its normal anatomical position, prevention of recurrence of its descent and without any complains to the patient.

A number of operations are done for the correction of vault prolapse of which abdominal sacropexy is the most scientific one. It is a durable and strong surgical correction for vaginal vault prolapse.<sup>(6)</sup>

**METHODS :**

We are presenting series of 10 cases of vaginal vault prolapse which reported to Sheth L. G. hospital over a period of 3 years from January 2010 to December 2012 in which abdominal sacropexy was planned. All Patients were between 55 to 75 years of age. They had their hysterectomy done before 10 to 15 years.

Pre-operatively on the operation table vaginal packing was done with roller pack to push vaginal vault above. Under spinal anesthesia abdomen was opened with a low transverse approach. Prominent vaginal vault held with two Allis forceps at both angles. Then vaginal pack was removed. Peritoneum overlying vaginal vault was dissected off anteriorly and bladder pushed downwards. The peritoneum was dissected posteriorly up to cul-de-sac also laterally on both sides to make vault free from peritoneum. When the vault reached at a higher level than sacral promontory, some upper part of vault was removed. For that nick kept at the center of vaginal vault and extended on both sides. Redundant and extra portion of vaginal vault were cut. Cut edges of vault were closed with vicryl no. 1-0 with continuous locking suture. Then peritoneum over sacral promontory was opened. Anterior spinal vessels were cauterized. A subperitoneal tunnel was created into cul-de-sac by passing long curved artery forceps in the direction of uterosacral ligament on right side upto vault. Now prolene

mesh of 10 × 6 cm folded and stitched at free ends with chromic catgut 2-0 to keep it folded. Then folded mesh pushed from behind space into the subperitoneal tunnel towards vault. At the vaginal vault the mesh was cut open in “fish-mouth” fashion and fixed at anterior and posterior aspect of vaginal vault with prolene no. 1-0 with interrupted sutures. Posterior end of mesh fixed to anterior longitudinal ligament with interrupted stitches of prolene no. 1-0. Peritoneum sutured on vault. Proper hemostasis achieved. Abdomen was closed in layers.

### RESULTS AND DISCUSSION :

All patients had vaginal vault prolapse with 3<sup>rd</sup> degree descent and no rectocele and cystocele.

#### TABLE – 1 : AGE OF PATIENTS

Table – 1 shows age of the patients. The mean age of patient was 65 years.

No. of patients	Age in years
4	55 to 65
6	66 to 75

#### TABLE – 2 : OPERATIVE RESULTS

Results	
(1) Average operative time	1.5 hours
(2) Intra and post operative Complications	Nil
(3) Post operative vaginal axis and Depth	Optimum

Table – 2 shows operative results. Average operative time was 1.5 hours. There were no intraoperative complications in form of bleeding or injury to any organ in our study. There was no difficulty in dissecting retroperitoneal space in any of the patients. In four patients because of very long vagina some part of vaginal wall cut and sutured as described in the method. All patients had uneventful postoperative period. This shows that abdominal sacropexy is a safe procedure if carried out with proper surgical skill. In all patients post operative vaginal depth was adequate and vaginal axis was optimum. No patient had dyspareunia or urinary complains in follow up at 6 months.

Comparing our results with some other studies with repair of vaginal vault prolapse shows good comparative result for abdominal sacropexy. The studies which have compared abdominal versus vaginal approach shows better and satisfactory results with abdominal sacropexy<sup>(5)</sup>.

Although recent studies have advocated laparoscopic sacropexy against abdominal or vaginal route, it can be done on selected patients by skilled surgeon in advanced operative set-up. In our study, most patients were above 65 years in whom spinal anesthesia was preferred in comparison to general anesthesia due to old age and associated medical conditions. Also, peri-operative complications such as bladder, rectal injury and hemorrhage in upto 5% of

cases and post-operative complications such as retropubic hematoma, perineal hematoma, hemorrhage and stapler induced peri-ostitis in upto 21% and re-operation in 4% patients is reported in laparoscopic sacropexy<sup>(2)</sup>.

### CONCLUSION :

Abdominal sacropexy is a safe and efficacious procedure, which is indicated in severe degree of prolapse of vault with significant loss of vaginal supports<sup>(1)</sup>. The abdominal route confers an advantage of consistent fixation of vaginal apex keeping a good depth of vagina and maintaining vaginal axis. If performed meticulously complications are minimal. Operative time and hospital stay are also optimum. Thus, abdominal sacropexy with prolene mesh is most valid technique in treatment of total vaginal vault prolapse<sup>(3, 4)</sup>.

### REFERENCES :

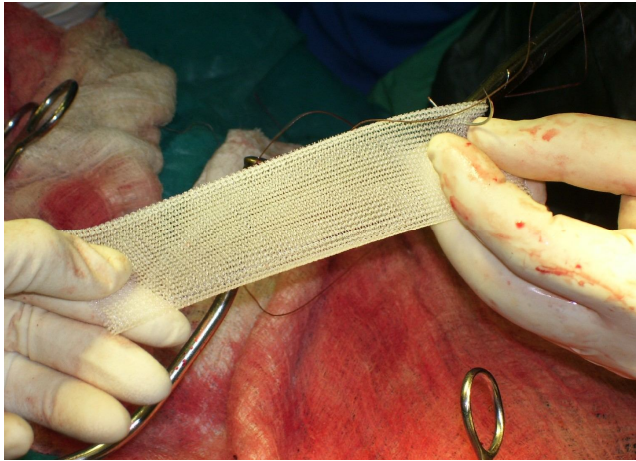
- (1) Anthony J. Woodruff (M.D.), Christopher C. Roth (M.D.), J. Christian Winters (M.D.), Abdominal sacral colpopexy : Surgical pearls and outcomes, Current Urology Reports 2007,8:399-404
- (2) Cosson, Rajabally, Bogaert, Queleu and Crepin. Laparoscopic Sacrocolpopexy, Hysterectomy and Burch Colposuspension: Feasibility and Short-Term Complications of 77 Procedures. Journal of Society of Laparoendoscopic Surgeons (JSLS) 2002 Apr-Jun; 6(2): 115-119
- (3) Diana M., Schettini M. Treatment of vaginal vault prolapse with abdominal sacral colpopexy using prolene mesh. Minerva Gynecology 1999;51(9)349-353
- (4) Diana M., Zoppe C., Mastrangeli B. Treatment of vaginal vault prolapse with abdominal sacral colpopexy using prolene mesh. American Journal of surgery 2000;179(2): 126-128
- (5) Maher C, Baessler K, Glazener CM, et al. Abdominal sacral colpopexy versus sacrospinous colpopexy (vaginal sacral colpopexy) for upper vaginal wall vault prolapse. Surgical management of pelvic organ prolapse in women. The Cochrane Library, Issue 4, 2005.
- (6) Parulekar Shashank V. (M.D., F.C.P.S., D.G.O., D.F.P.), Shah Jatin P. (M.D., D.G.O.). Abdominal Sacropexy. Photographic atlas of gynecologic and obstetric operative techniques, Bhalani Vinod S., Bhalani Publishing House, Mumbai. 1992, ISBN 81-85578-02-8. 151 – 154



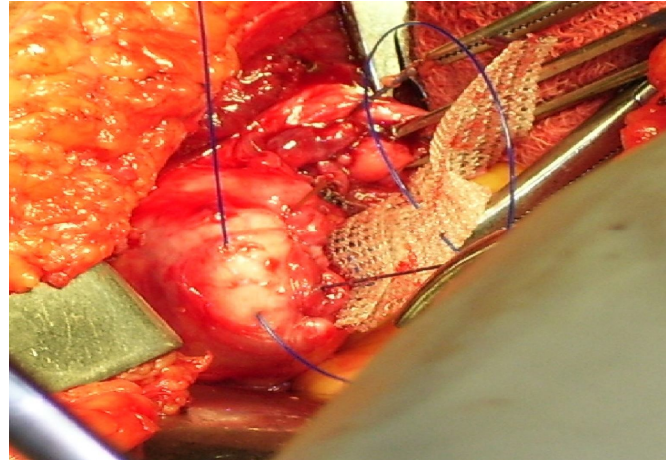
## LEGENDS OF PHOTOGRAPHS

### Photograph – 1

Folding of mesh with stitches



### Photograph – 2



## (18) IS AGE OF ONSET OF MYOCARDIAL INFARCTION AND SERUM CHOLESTEROL LEVEL RELATED TO SMOKING STATUS ?

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### **Abstract**

**Introduction:** Smoking is a habit indulged in different forms almost all over the world. It is done solely for personal pleasure and satisfaction, as a status symbol for some and as a method of relieving anxiety for others. The background leading to the present study was that cigarette smokers are known to be more prone to various cardiorespiratory diseases.

**Objective:** The study was done with an intention to find out the effect of smoking on serum cholesterol levels and the age of onset of Acute Myocardial Infarction (AMI).

**Method:** The present study was conducted on 20 smokers and 20 Non-smokers. Patients admitted in the Cardiology unit for AMI were checked out for their cholesterol levels and age of onset of AMI and further compared with their smoking history.

**Results:** On analysis it was revealed that smokers had a significant ( $p > 0.05$ ) higher serum total cholesterol level than non-smokers. Further it was also found that smokers presented with AMI and Ischaemic heart disease at a much earlier age than smokers.

**Conclusion:** Smoking habit thus shown to be a potent contributor to cardiovascular disease should definitely be curbed at all possible levels.

### Key words

Smokers, cigarettes, myocardial infarction, serum cholesterol

### Introduction

The custom of smoking dried tobacco leaves spread from America to the rest of the world after European colonization began in the sixteenth century. According to subjective experiences the effects of smoking are soothing and tranquilizing and during 'appropriate' circumstances there also exists a stimulant action. Smoking also is known to give a sense of well being. It is done solely for personal pleasure and satisfaction. For some it is also a status symbol while for others it is also a method of relieving anxiety.

But worldwide smoking has come to be recognized as a formidable risk factor in the morbidity and mortality of Coronary heart Disease (CHD) and Ischaemic Heart Disease (IHD). Even in Indians IHD is becoming an alarming cause of death. Some deaths occur suddenly as a result of an acute coronary occlusion and/or of myocardial fibrillation, whereas others occur slowly over a period of weeks to years as a result of progressive weakening of the heart. Nicotine and carbonmonoxide which are released in the blood due to smoking seem to play a major role in producing the deleterious effects like atherosclerosis, damaged endothelium, tissue anoxemia, vasospasm and enhanced platelet aggregation in the blood vessels of smokers. Even the adverse effects of smoking and its relationship with plasma lipids and lipoproteins have come to light recently and is a subject of research in many studies.

In spite of so much work done and worldwide propagation about the ill effects of smoking individuals at a very tender age are regularly falling prey to this lethal habit of smoking. Due to this early habituation of smoking the period of 'Smoking Years' is on the rise and thereby probably affecting the age of incidence of CHD and IHD.

Taking into account the above mentioned effects of smoking the present study was thus conducted with a motive to investigate whether the Age of onset of myocardial Infarction and serum cholesterol level are related to the smoking status.

### Materials and Methods

The present study was conducted on the patients with AMI admitted at the Cardiology

Department of Grant Govt. Medical College and J.J.Hospital Mumbai. Subjects which were included in the study were males and consisted of 20 smokers and 20 non smokers. The Non-Smokers were patients who had never smoked or used tobacco in any form. The Smokers were patients with a history of Heavy smoking i.e. smoking more than 15 cigarettes per day.

### **Criteria for Diagnosis of AMI**

The diagnosis of Acute Myocardial Infarction was established by the clinicians on the basis of the presence of at least two of the following criteria:

- 1) Characteristic Chest pain
- 2) ECG changes with appearance of Q waves( transmural infarction)
- 3) Characteristic elevation of Serum aspartate aminotransferase or serum creatinekinase.
- 4) Non-Transmural infarction was diagnosed by typical ST segments and T wave changes accompanied by atleast criteria No. 3

**The subjects for the present study were selected on the basis of the following Criteria:**

#### **Inclusion Criteria :**

**Age :** Male patients in the age range of 35-75 years after noting down their age have been included.

**Sex :** Only male patients were considered for the present study to avoid the variation in the values due to gender differences.

**Socioeconomic Status :** all patients chosen belonged to the middle socioeconomic status.

#### **Exclusion Criteria :**

Following diseases were looked for and excluded to avoid their complications affecting the present study.

- 1) **Diabetes Mellitus :** Patients whose Fasting Blood Sugar (FBS) were less than 120 mg/dl were chosen.
- 2) **Hypertension :** Men were excluded if they had hypertension or were receiving antihypertensive medication.
- 3) **Hypothyroidism :** Serum T3, T4 and TSH levels were seen to exclude hypothyroidism.
- 4) **Obstructive Liver Disease :** This was excluded on the basis of serum bilirubin (Total and Direct).
- 5) **Nephrotic syndrome :** This was excluded on the basis of the clinical judgement and acumen of the clinicians.
- 6) **Obesity :** Patients who were not overweight for their height and age were selected.

Fasting blood samples of all the forty subjects were collected and total serum cholesterol was estimated using the standard CE-CO PAP Enzymatic End point Method.(1,2).Total Cholesterol Values were measured in mgs/dl.

All the results thus obtained by Laboratory analysis were noted down and used for subsequent statistical analysis.

## Statistical analysis

Results obtained in the study are expressed as mean  $\pm$  SD. The statistical analysis of the data obtained for the smokers and non smokers groups were done using chi-square test and ANOVA. p values  $<0.05$  was considered significant and those with p value  $< 0.001$  was considered highly significant. For the analysis of the data Microsoft Excel software as well as 'Graphpad instat, version 03, sandiago, california' were used.

## Observation and results

Serum Total cholesterol levels and the age of onset of AMI were compared in the Smokers and Non-Smokers. When Serum Total cholesterol levels in the smokers and non smokers were analyzed it was revealed that the smokers had a highly significant ( $p<0.001$ ) increase in the serum total cholesterol levels as compared to those of the Non-smokers. Similarly when Age of onset of AMI in the smokers and the Non Smokers was compared it was found that the smokers had a highly significant ( $p<0.001$ ) earlier onset of AMI as compared to those of the Non Smokers.

**Table showing the Serum Total Cholesterol levels and the Age of onset of AMI in the Smokers and Non Smokers.**

	<b>Serum Total Cholesterol levels in mg/dl</b>	<b>Age of onset of AMI in Years</b>
Non Smokers	167.50 $\pm$ 12.67	66.00 $\pm$ 9.98
Smokers	227.50 $\pm$ 13.87	43.40 $\pm$ 6.00
Significance	p $<$ 0.001(H.S.)#	p $<$ 0.001(H.S.)##

Footnote – all the values in the table are expressed as mean  $\pm$  SD

H.S. - Highly Significant

# comparison of Serum total cholesterol levels between smokers and non smokers.

## comparison of Age of onset of AMI in smokers and non smokers.

## Discussion

Coronary Heart Disease appears to be the product of a faulty life style and one of the major components of these deleterious living habits is cigarette smoking. Smoking has shown to cause a transient increase in the adhesiveness of platelets, accelerates the heart rate and makes the myocardium more prone to ventricular fibrillation. At the same time there is reduced oxygen capacity of the blood because of the build of carboxyhemoglobin, which is further aggravated by impaired tissue utilization of oxygen at the myoglobin level.(3) Also Nicotine and carbonmonoxide which are released in the blood due to smoking seem to play a major role in producing the lethal effects like atherosclerosis, damaged endothelium, tissue anoxemia, vasospasm and enhanced platelet aggregation in the blood vessels of smokers. (4,5)These and other atherogenic effects of smoking has come to be recognized as a formidable risk factor in the morbidity and mortality of Coronary heart Disease(CHD) and Ischaemic Heart Disease(IHD).

Studies done by different researchers have highlighted the effects of smoking on IHD, CHD and Myocardial disease related mortality and morbidity.

Doyle et al in 1983(6) had shown cigarette smoking to be a risk factor for myocardial infarction and also showed the effect of greatly increasing the probability of acute myocardial events in patients with hypercholesterolemia.

Oscar Auerbach et al in 1976(7) had shown in studies of autopsies an increase of atherosclerotic lesions in the coronary arteries of cigarette smokers. These lesions did not occur in those who never smoked. However moderate and advanced thickening was observed in 98.6 percent of those individuals who smoked two or more packs per day.

Bansal R D et al in their 1970(8) study carried out in the Cardiology clinic at AIIMS, New Delhi had implicated cigarette smoking to be a risk factor for CHD patients.

Studies done by Jayant K et in 1983(9) showed that cigarette smokers had a 2.8 fold risk of myocardial infarction than the non smokers.

Two separate studies done by Jindal S K et al in 1982(10) and Bhowan A S et al in 1969 (11) have shown carboxyhemoglobin to be higher in smokers compared to non smokers which is injurious to the Myocardium considering the above mentioned harmful effects of carboxyhemoglobin on the vascular endothelium and platelet aggregation.

In 1985 Tamsin Lisa et al (12) had shown in their study that smoking is a risk factor for cardiovascular disease and may contribute to the occurrence of AMI at a younger age. Smokers as compared to Non smokers were 10 years younger as far as Cardiovascular risks were concerned.

The Data from the study done by Doll Richard et al in 1976(13) has also shown a twofold increase of death rate among young smokers as compared to non smokers.

Throughout the world individuals at a very young age are increasingly falling prey to this lethal habit of smoking, thus affecting the age of incidence of CHD and IHD. The 'Smoking Years' or the number of years that an individual actively smokes is also on the rise because of this early introduction to smoking in these youngsters. Our study has shown that the persons who smoke more than 15 cigarettes per day present with IHD at a much earlier age than the Non-smokers.

Some studies done by different researchers have also highlighted the effects of smoking on Serum total cholesterol levels.

Hjermann Ingvar et al in 1976(14) showed that in cigarette smokers the serum cholesterol increases with increasing daily number of cigarettes.

Cheryl Brischetto et al in 1985(5) had observed a higher level of serum Total cholesterol and serum triglycerides in heavy cigarette smokers.

Karvonen et al in 1959 (15) had reported from his studies made in Finland that overall mean cholesterol level of smokers was significantly higher than the non smokers.

Rastogi and Srivastava in their study in 1989 (16) had reported that the mean levels of serum cholesterol and triglycerides were significantly higher in smokers.

Similarly a study done by Casanova and Lapetra in 1992 (17) which had included cadets from military academy in Zaragoza had shown higher levels of serum triglycerides and total cholesterol in smokers.

In accordance with the above studies our study too revealed significantly higher total cholesterol levels than those of the non smokers.

But contrary to the above findings a study done by Jansen D.F. et al in 1995(18) showed that Smoking was not associated with total cholesterol or with HDL cholesterol levels.

Trying to relate the effects of smoking on serum cholesterol, it is a known fact that among the effects of smoking there is an alteration of lipid metabolism through a increase in lipolysis, insulin resistance and tissue lipotoxicity. (19)

Nicotine in cigarettes stimulates sympathetic adrenal system leading to increased secretion of catecholamine resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA) which further results in increased secretion of hepatic FFA and hepatic triglycerides in the blood stream. (20,22)

Dyslipidemia is a well established risk factor for the development of CAD. (21,22) Our study thus demonstrated the presence of dyslipidemia in the form of an increase in Total Cholesterol in the smokers, and thereby pointing towards the risk of CAD.

A ray of hope for smokers could lie in the fact that Quitting smoking to a certain extent could still reduce the incidence of CAD and IHD in them. This could be seen from certain studies like that done by Hijermann I et al in 1981(23) in which a 5 year randomized trial showed lowering of serum lipids after the cessation of smoking thus lowering the incidence of CAD. This study also showed a decreased incidence of myocardial infarction and sudden death after stopping the smoking habit. World health Organisation(WHO) also as a health perspective has made several recommendations about how tobacco should be controlled in order to reduce the disease, disability and premature death in the form of stopping promotion of tobacco products, public education and public information, reducing emission levels of toxic components and controlling smoking in public places.(24)

## **Conclusion and summary**

The present study thus highlights the redundant effects of smoking in terms of increased incidence of cardiovascular disease at an earlier age and increased total cholesterol levels in the smokers as compared to the non smokers. Hence cigarette smoking is so powerful a contributor, so highly prevalent and so potentially correctable that it deserves the highest priority among preventive measures to control cardiovascular disease.

## **References**

- 1) CC Allain, LS Poon, GSG Chan, W. Richmond, PC Fu; Clin. Chem, 20 (1974), pp. 470–475
- 2) Chatterjea, M. N., & Shinde, R. (2002). Textbook of medical biochemistry.
- 3) Kannel, W. B. (1981). Update on the role of cigarette smoking in coronary artery disease. *American heart journal*, 101(3), 319.
- 4) McKenna, W. J., Chew, C. Y., & Oakley, C. M. (1980). Myocardial infarction with normal coronary angiogram. Possible mechanism of smoking risk in coronary artery disease. *British heart journal*, 43(5), 493-498

- 5) Brischetto, C. S., Connor, W. E., Connor, S. L., & Matarazzo, J. D. (1983). Plasma lipid and lipoprotein profiles of cigarette smokers from randomly selected families: enhancement of hyperlipidemia and depression of high-density lipoprotein. *The American journal of cardiology*, 52(7), 675-680.
- 6) Doyle, J. T., Dawber, T. R., Kannel, W. B., Kinch, S. H., & Kahn, H. A. (1964). The relationship of cigarette smoking to coronary heart disease. *JAMA: the journal of the American Medical Association*, 190(10), 886-890.
- 7) Auerbach, O., Carter, H. W., Garfinkel, L., & Hammond, E. C. (1976). Cigarette smoking and coronary artery disease. A macroscopic and microscopic study. *CHEST Journal*, 70 (6), 697-705.
- 8) Bansal, R. D., Chablani, T. D., & Gulati, P. V. (1970). An epidemiological study of ischaemic heart disease in patients attending the cardiology clinic at the AIIMS, New Delhi. *Indian medical journal*, 64(4), 84-92.
- 9) Jayanta, K., Gulati, S. S., & Sanghvi, L. D. (1983). Tobacco use in relation to coronary heart disease. A case-control study in Bombay, India. *World smoking and health*, 8, 15-8.
- 10) Jindal S.K., Malik S.K., Bange N. (1982) Blood carboxy hemoglobin in cigarette and bidi smokers. *Indian J. Chest Dis. And allied Sci.* (special number on smoking and health), 203-207
- 11) Bhowan A.S., Maitrya B.B., Hag I.U. (1969) Blood carboxy haemoglobin level in Bidi smokers. *Indian J. Med.* 57, 1313-1315
- 12) Tamsin Lisa, K., Gilpin, E., Ahnve, S., Henning, H., & Ross, J. (1985). Smoking status at the time of acute myocardial infarction and subsequent prognosis. *American heart journal*, 110 (3), 535-541.
- 13) Doll, R., & Peto, R. (1976). Mortality in relation to smoking: 20 years' observations on male British doctors. *British medical journal*, 2(6051), 1525.
- 14) Hjerman, I., Helgeland, A., Lund-Larsen, I. H. P. G., & Leren, P. (1976). The Intercorrelation of Serum Cholesterol, Cigarette Smoking and Body Weight. *Acta Medica Scandinavica*, 200 (1-6), 479-485.
- 15) Karvonen, M., Orma, E., Keys, A., Fidanza, F., & Brozek, J. (1959). Cigarette smoking, serum-cholesterol, blood-pressure, and body fatness observations in Finland. *The Lancet*, 273(7071), 492-494.
- 16) Rastogi, R., Shrivastava, S. S., Mehrotra, T. N., Singh, V. S., & Gupta, M. K. (1989). Lipid profile in smokers. *The Journal of the Association of Physicians of India*, 37(12), 764.
- 17) Casonova J. A., Lapetra A. (1992) Tobacco, Physical Exercise and lipid profile, *Eur. Heart J.* 13 (4):440-445
- 18) Jansen, D. F., Nedeljkovic, S., Feskens, E. J., Ostojic, M. C., Grujic, M. Z., Bloemberg, B. P., & Kromhout, D. (1995). Coffee consumption, alcohol use, and cigarette smoking as Countries Study. *Arteriosclerosis, thrombosis, and vascular biology*, 15(11), 1793-1797.
- 19) Gastaldelli, A., Folli, F., & Maffei, S. (2010). Impact of tobacco smoking on lipid metabolism, body weight and cardiometabolic risk. *Current pharmaceutical design*, 16(23), 2526-2530.
- 20) Neki, N. S. (2002). Lipid profile in chronic smokers - A clinical Study. *JACM*, 3(1), 51-4.

- 21) Penalva, R. A., Huoya, M. D. O., Correia, L. C. L., Feitosa, G. S., & Ladeia, A. M. T. (2008). Lipid profile and intensity of atherosclerosis disease in acute coronary syndrome. *Arquivos Brasileiros de Cardiologia*, 90(1), 24-30.
- 22) Kshitish Kumar Kshitiz\*1, Ram Binay Sinha2 And Jayashree Bhattacharjee1 A Study Of Effects Of Smoking On Lipid And Vitamin C Metabolism. A Pilot Study In Central Bihar. (2010) *International Journal of Pharma and Bio Sciences*,1(4) 106-113
- 23) Hjermann, I., Velve, B. K., Holme, I., & Leren, P. (1981). Effect of diet and smoking intervention on the incidence of coronary heart disease. Report from the Oslo Study Group of a randomised trial in healthy men. *Lancet*, 2(8259), 1303
- 24) Notani, P.N., Jayant, K. and Sanghvi, L.D. (1989) Assessment of morbidity and mortality due to tobacco usage in India. In Sanghvi, L.D. and Notani, P. (eds) *Tobacco and Health: The Indian Scene*. UICC Publication, Tata Memorial Centre, Bombay, India, pp. 63–78.

## **Message of World health day 7th April 2013 by SEAR of WHO on High Blood Pressure**

World Health Day is celebrated on 7 April to mark the anniversary of the founding of WHO in 1948. Each year a theme is selected for World Health Day that highlights a priority area of public health concern in the world.

The theme for 2013 is High blood pressure.

High blood pressure increases the risk of heart attacks, strokes and kidney failure. However, hypertension is treatable and preventable.

High blood pressure – also known as Hypertension – increases the risk of heart attacks, strokes and kidney failure. Uncontrolled hypertension can also cause blindness, irregularities of the heartbeat and heart failure. The risk of developing these complications is higher in the presence of other cardiovascular risk factors such as diabetes. One in three adults has high blood pressure in WHO's South-East Asia Region. Nearly 1.5 million people die due to high blood pressure every year making it a leading risk factor for mortality in the Region.

However, hypertension is preventable and treatable. In some developed countries, prevention and treatment of hypertension, together with other cardiovascular risk factors, has brought about a reduction in deaths from heart disease. The risk of developing hypertension can be reduced by:



- reducing salt intake
- eating a balanced diet
- avoiding harmful use of alcohol
- regular physical activity
- maintaining a healthy body weight
- avoiding tobacco use

### **Goals :**

Greater awareness, healthy behaviours, improved detection and enabling environments. The ultimate goal of World Health Day 2013 is to reduce heart attacks and strokes. Specific objectives of the campaign are:

- to raise awareness of the causes and consequences of high blood pressure,
- to encourage people to change behaviours that can lead to hypertension,
- to persuade adults to have their blood pressure checked regularly,
- to increase the number of health facilities offering blood pressure checks and
- to incite national and local authorities to create enabling environments for healthy behaviours.

### **The problem**

High blood pressure (also known as raised blood pressure or hypertension) can lead to heart attack, stroke and other serious health problems. It affects more than one in three adults and leads to more than nine million deaths worldwide every year. High blood pressure can also cause kidney failure, blindness, rupture of blood vessels and brain impairment.

Many people do not know that they have high blood pressure because it does not always cause symptoms. Even though it is easily diagnosed and treated, many people do not have access to basic health services, particularly in low- and middle-income countries.

### **The solution**

High blood pressure is both preventable and treatable.

Controlling high blood pressure, together with other risk factors, is the main way to prevent heart attack and stroke.

The risk of developing high blood pressure can be minimized by: cutting down on salt; eating a balanced diet; avoiding harmful use of alcohol; getting regular exercise; and avoiding tobacco use.

For many people, lifestyle changes are sufficient to control blood pressure. For others, medication is required. Inexpensive medication exists, which is effective when taken as prescribed.

It is essential that detection and control of high blood pressure (measurement, health advice and treatment), are coupled with simultaneous reduction of other risk factors that cause heart attacks and strokes, such as diabetes and tobacco use. They should be core elements of

primary health care in all countries, and integral to efforts to reduce the growing burden of noncommunicable diseases.

Civil society has an important role to play in helping to address high blood pressure.

Industry can contribute to the solution, for example, by reducing salt in processed food and making essential diagnostics and medicines more affordable.

Early detection is key; all adults should know their blood pressure.