

## **Influence of Preksha Meditation on Blood Profile of Adults**

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### **Abstract**

**Objective:** The present study was envisaged to investigate whether Preksha Meditation practice yields any positive change in various components of blood chemistry there by promoting the physiological health state. **Subjects:** 40 healthy adults in the age group of 25 to 30 years were selected on random basis and divided into two groups, each containing 20 subjects. The first group of 20 subjects named 'experimental group' was exposed to selected Preksha Meditation practice module for 50 minutes once a day for 90 days and second group of 20 subjects, named 'control group' live their routine life. **Method:** On the first day of experiment the subjects Arterial blood pressure (BP), serum glucose, ESR hemoglobin RBC Count and Lipid profile were recorded and same repeated after 90 days. **Results:** the experimental group of subjects show an improvement in all the variables but no such difference was there in control group. **Conclusions:** Preksha Meditation practice yields positive change in above variables of blood there by promoting the physiological health state.

**Keywords:** Preksha Meditation, Blood Profile, Lipid Profile, Physiological health.

### **Introduction**

Meditation is a word that has been used in variety of ways but all of them define it as thinking contemplation, concentrating mind on a object, paying attention etc. but in the tenets propounded by Lord Mahaveer "*Perceive and know*" is given more prominence because perception is strictly concerned with the phenomenon of the present, neither past nor future. He stated "*Sampikkhae appagamappaenam*" means 'see you thyself' or perceive and realize yourself, which later becomes the principle of the Jain yoga tradition, and formulated as Preksha Meditation by Late Acharya Tulsi and Acharya Mahaprajna

The word *Preksha* is derived from the root *iksa*, which means 'to see'. When the prefix '*pra*' is added, it becomes *pra + iksa = preksa*, which means to 'perceive carefully and profoundly'<sup>1</sup>. Here 'seeing' does not mean external vision, but careful concentration on subtle consciousness by mental insight. *Preksha Dhyana* is the system of meditation engaging one's mind fully in the perception of subtle internal and innate phenomena of consciousness.

*Kayotsarga* (total relaxation with self awareness), *Deergha Swas Preksha* (Perception of Deep Breathing) and *Jyotikendra Preksha* (Perception of center of enlightenment) are integral constituents of *Preksha* Meditation. *Kayotsarga* is practiced to counteract the ill effects of various stimuli causing stress, as the stress management is one of the most important lessons for remaining healthy. It helps modulating the metabolic rate in the body and brings back normalcy in various biochemical profiles which are essential for maintaining good health and general well being. *Deergha Swasa Preksha* provides a continual replenishment of the oxygen in the lungs, drawing in fresh air and expelling waste gases which ultimately maintains the normal metabolic rate at cellular level. *Jyotikendra Preksha* is the perception of a psychic center represented by one of the endocrine glands, the Pineal Gland. Perceiving the *Jyotikendra*, the state of balanced functional coordination is achieved in two control systems of the body, namely Nervous system and Endocrine system. There are experimental evidences which indicate that pineal hormones inhibit ACTH secretion and thus indirectly help to regulate the secretion of hormones of another endocrine gland, the Adrenal gland which are responsible for various metabolic activities<sup>2</sup>.

Blood is a vital fluid in the body. Basically it is transport medium of various essential chemical compounds and gases. At the same time its own constituent cellular and liquid components are also pivotal in modulating the detrimental actions and reactions of health and well being. Red Blood Corpuscles (RBC), Haemoglobin (Hb), Erythrocyte Sedimentation Rate (ESR), Glucose and Lipid Profile are such components. In the present study it was envisaged to investigate whether *Preksha* Meditation practice yields any positive change in these components there by promoting the physiological health state<sup>3</sup>.

Joshi<sup>4</sup> conducted a study to find out the impact of *Kapalbhati*, *Vaman* and *Bhramari* *Pranayama* on college going students and observed that there was a significant relationship between the practice of *Kapalbhati*, *Vaman* and *Bhramari* on Hemoglobin (increase from 12.2 gm% to 13.10 gm %), E.S.R. (decrease from 9.5 to 3.45), F.V.C. (increase 65.05% to 88.02%), along with strong physical and mental well being. Jevning<sup>5</sup> reported a fundamental change in cellular metabolism, reduced glucose metabolism in red blood cells after the practice of *Transcendental Meditation*. Smith et al<sup>6</sup> in their study on *Transcendental Meditation (TM)* program on adult subjects found a lower ESR and concluded that it is an indicator of less

serious illness and slower aging. Jevning et al<sup>7</sup> in their study found a fundamental change in cellular metabolism, reduced glucose metabolism in red blood cells, normal arterial oxygen and carbon dioxide partial pressures, reduction in biochemical index of stress and reduced spontaneous skin resistance responses in the subjects practicing yoga.

With reference to Preksha Meditation it was found in a study that lipid profile of both patients of diabetes<sup>8</sup> and normal healthy subjects<sup>9,10</sup> show positive improvements. This encourages us to conduct the present study.

### **Material and Methods**

40 healthy adults in the age group of 25 to 30 years were selected on random basis and divided into two groups, each containing 20 subjects. Best of possible efforts were made to include the subjects having similar socioeconomic status. However, qualifications and professional uniformity could not be taken into consideration because of local geographical constraints. The first group of 20 subjects named 'experimental group' was exposed to selected Preksha Meditation practice module for 50 minutes once a day for 90 days and second group of 20 subjects, named 'control group' live their routine life and no intervention was given to them. Experimental intervention module was comprised of following Preksha Meditation components:

- |                            |         |
|----------------------------|---------|
| i. Kayotsarga              | 10 min. |
| ii. Deergha swansa preksha | 20 min. |
| iii. Jyotikendra Preksha   | 20 min. |

On the first day of experiment the subjects reported at our laboratory in the morning after overnight fast. The laboratory temperature was maintained  $27^0 \pm 2^0\text{C}$ . All the measurements were taken in the resting supine position. All the tests were again carried out after 90 days by using same methods in both control and experimental group of subjects.

In this study both inter-group and intra-group comparisons were made with the aim to evaluate the net effect of Preksha Meditation practice module vis-à-vis normal activity. One tailed Sadler's 'A' test and student's 't' test were applied for intra and inter group comparison respectively and graded significance level was denoted as  $P \leq 0.05$  (\*), which was taken from probability significant task.

## **Result**

### **Blood Glucose**

The blood glucose has shown a similar range of values in both control and experimental group of subject at 0 day of observation which was quite obvious because none of the subjects of these two groups were given any specific treatment. The basal mean fasting blood sugar levels were  $81.3529 \pm 4.73467$  and  $81.1176 \pm 4.66947$  mg/dl in control group and experimental group of subjects respectively. After 90 days of the experiment a sharp decline was noticed in the fasting blood sugar in the experimental group of subjects where the mean value was reported to be  $79.7059 \pm 3.92760$  mg/dl (Figure 1). The difference was statistically significant. However, in the control group the mean blood sugar at 90 days was  $80.7941 \pm 4.09567$  mg/dl, which was not statistically significant (Table 1). Both groups are at homogeneity as no significant difference was there at pre stage, but after 90 days a significant difference was there in mean values of both groups.

### **Hemoglobin**

The basal values of hemoglobin in blood of both control and experimental group were  $12.4412 \pm 1.07847$  gm/dl and  $12.3824 \pm 0.77907$  gm/dl respectively. The mean value of hemoglobin in experimental group slightly increases after 90 days of practice of Preksha Meditation and the change was statistically significant. In control group of subjects the mean values remain almost in similar range without and statistical significance (Table 1). Inter group comparison of the mean values of hemoglobin in experimental and control group of subjects at different phases shown in Table 2 has not shown any significant change was there at 0 day as well as after 90 days.

### **Erythrocyte Sedimentation Rate (ESR)**

ESR mean values of the both control and experimental group at 0 day were  $8.3500 \pm 1.05061$  mm/hr and  $8.4294 \pm 1.04433$  mm/hr. After 90 days of Preksha Meditation intervention the mean value was recorded to be decreased to  $8.2000 \pm 0.95759$  mm/hr, in experimental group and the difference was statistically significant. In control group of subjects no such decline was noticed (Table 1). After 90 days a significant difference was observed in mean values of ESR in between both groups (Table 2).

### **Red Blood Corpuscle Count (RBC count)**

At the onset of the experiment the RBC count of the experimental group of subjects was  $5.282 \pm 0.3973$  million/mm<sup>3</sup> and it increased up to  $5.317 \pm 0.4253$  after 90 days

of Preksha Meditation practice (Figure 1). This change was statistically significant at  $p \leq 0.05$  level. But the same in control group of subjects was  $5.138 \pm .4397$  at onset and came to be  $5.135 \pm 0.3922$  at 90 days, without statistical insignificance (Table 1). Again after 90 days a significant difference was noticed in mean values of RBC count in both groups.

#### Very Low Density Lipoprotein

The mean serum VLDL level in control group of subjects was estimated to be  $34.8059 \pm 2.20604$  and  $34.9588 \pm 2.08822$  mg/dl at 0 and 90 respectively. The mean serum VLDL values in experimental group of subjects were found to be  $34.7059 \pm 1.96961$  and  $34.3824 \pm 1.88864$  mg/dl at 0 and 90days respectively, showing a significant decline at 90 days when compared with the basal values at 0 day (Figure 1). Both groups were at homogeneity as no significant difference was there at pre stage (Table 2), Follow up schedule of 90 days has shown a significant difference in mean values of VLDL in both groups.

Table 1: Intra - group comparison of dependent variables

| Sr.no | Name of Parameter                    | Experimental Group  |                      | 'A'                | Control Group       |                      | 'A'                 |
|-------|--------------------------------------|---------------------|----------------------|--------------------|---------------------|----------------------|---------------------|
|       |                                      | Pre (Mean $\pm$ SD) | Post (Mean $\pm$ SD) |                    | Pre (Mean $\pm$ SD) | Post (Mean $\pm$ SD) |                     |
| 1     | RBC count (million/mm <sup>3</sup> ) | 5.28 $\pm$ 0.39     | 5.31 $\pm$ 0.42      | 0.06 <sup>NS</sup> | 5.13 $\pm$ 0.43     | 5.13 $\pm$ 0.39      | 6.11 <sup>NS</sup>  |
| 2     | Haemoglobin (gm/dl)                  | 12.38 $\pm$ 0.95    | 12.91 $\pm$ 0.86     | 0.07 <sup>NS</sup> | 12.44 $\pm$ 1.07    | 12.55 $\pm$ 0.89     | 1.12 <sup>NS</sup>  |
| 3     | Glucose (mg/dl)                      | 81.11 $\pm$ 4.66    | 79.7 $\pm$ 3.9       | 0.06 <sup>NS</sup> | 81.35 $\pm$ 4.73    | 80.79 $\pm$ 4.09     | 0.52 <sup>NS</sup>  |
| 4     | ESR (mm/Hr)                          | 8.4 $\pm$ 1.04      | 8.2 $\pm$ 0.95       | 0.05 <sup>NS</sup> | 8.35 $\pm$ 1.05     | 8.4 $\pm$ 1.07       | 2.16 <sup>NS</sup>  |
| 5     | TC (mg/dl)                           | 183.9 $\pm$ 5.07    | 180.6 $\pm$ 4.76     | 0.05 <sup>NS</sup> | 181.9 $\pm$ 6.34    | 182.1 $\pm$ 4.35     | 10.25 <sup>NS</sup> |
| 6     | LDL (mg/dl)                          | 110.11 $\pm$ 4.46   | 107.55 $\pm$ 4.25    | 0.05 <sup>NS</sup> | 109.04 $\pm$ 5.29   | 108.92 $\pm$ 4.55    | 26.38 <sup>NS</sup> |
| 7     | Tg (mg/dl)                           | 173.52 $\pm$ 9.84   | 171.91 $\pm$ 9.44    | 0.09 <sup>NS</sup> | 174.02 $\pm$ 11.03  | 174.79 $\pm$ 10.44   | 0.72 <sup>NS</sup>  |
| 8     | VLDL (mg/dl)                         | 34.71 $\pm$ 1.96    | 34.38 $\pm$ 1.88     | 0.09 <sup>NS</sup> | 34.81 $\pm$ 2.21    | 34.95 $\pm$ 2.1      | 0.72 <sup>NS</sup>  |
| 9     | HDL (mg/dl)                          | 38.17 $\pm$ 2.52    | 38.71 $\pm$ 2.32     | 0.08 <sup>NS</sup> | 38.08 $\pm$ 2.82    | 38.29 $\pm$ 2.26     | 0.95 <sup>NS</sup>  |

<sup>NS</sup> – Not Significant, \* -  $p \leq 0.05$

Figure 1: Graph showing intra group comparison of experimental group

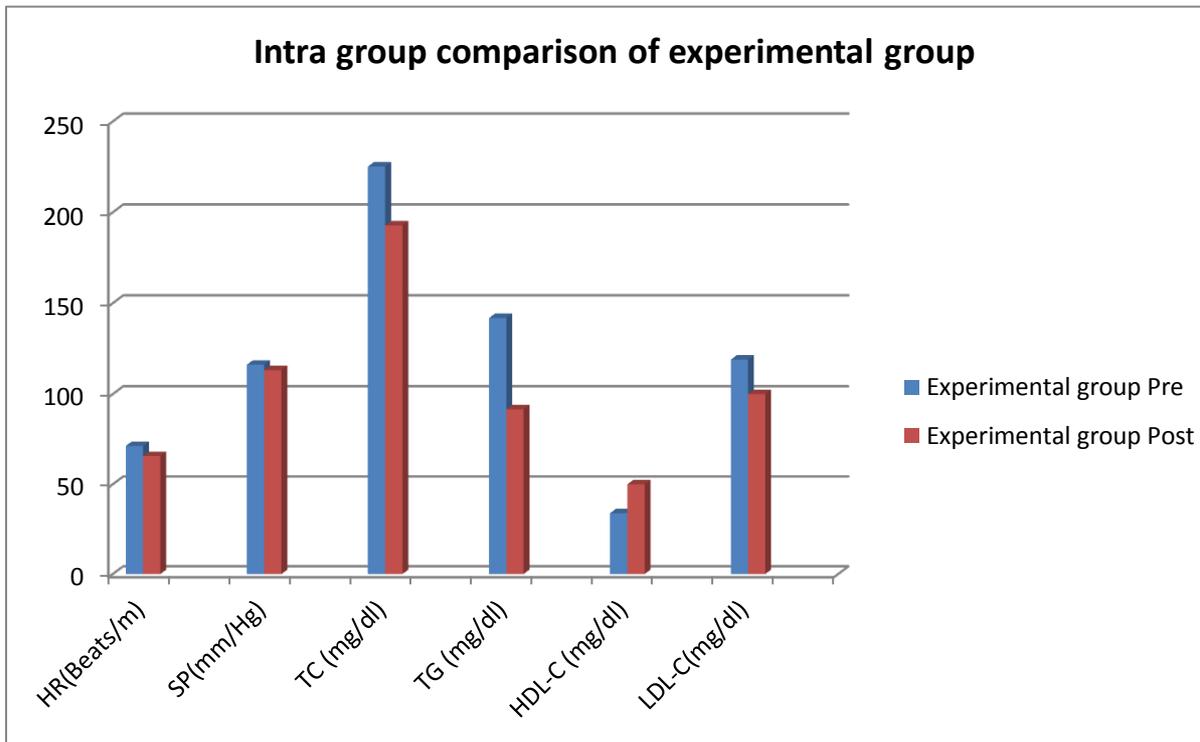


Figure 2: Graph showing Intra group comparison of control group

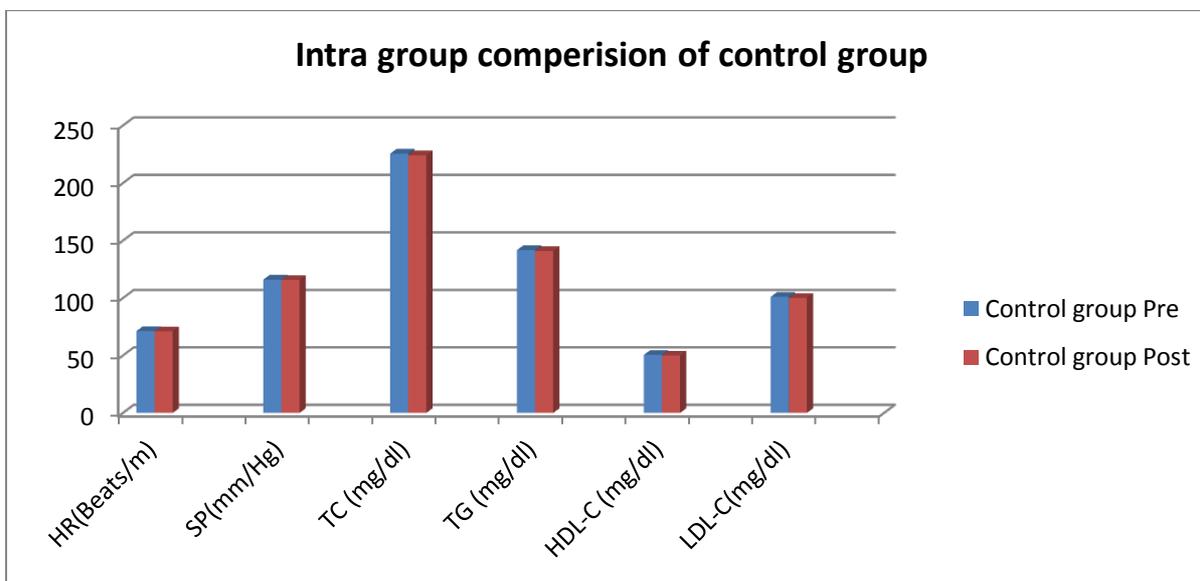


Table 2: Inter group comparison of depending variables

| Sr. No. | Parameter                            | Group        | Duration | Mean Value | ±SD      | t            |
|---------|--------------------------------------|--------------|----------|------------|----------|--------------|
| 1.      | RBC Count (million/mm <sup>3</sup> ) | Control      | 0 day    | 5.138      | ±0.4397  | -1.436<br>NS |
|         |                                      | Experimental | 0 day    | 5.282      | ±0.3973  |              |
|         |                                      | Control      | 90 days  | 5.135      | ±0.3922  | -4.619*      |
|         |                                      | Experimental | 90days   | 5.317      | ±0.4253  |              |
| 2.      | Haemoglobin (gm/dl)                  | Control      | 0 day    | 12.4412    | ±1.07847 | 0.627<br>NS  |
|         |                                      | Experimental | 0 day    | 12.3824    | ±0.95393 |              |
|         |                                      | Control      | 90 days  | 12.5588    | ±0.89413 | -0.627<br>NS |
|         |                                      | Experimental | 90days   | 12.6176    | ±0.77907 |              |
| 3.      | Glucose (mg/dl)                      | Control      | 0 day    | 81.3529    | ±4.73467 | 0.686<br>NS  |
|         |                                      | Experimental | 0 day    | 81.1176    | ±4.66947 |              |
|         |                                      | Control      | 90 days  | 80.7941    | ±4.09567 | 3.960*       |
|         |                                      | Experimental | 90days   | 79.7059    | ±3.92760 |              |
| 4.      | ESR (mm/Hr)                          | Control      | 0 day    | 8.3500     | ±1.05061 | -1.099<br>NS |
|         |                                      | Experimental | 0 day    | 8.4294     | ±1.04433 |              |
|         |                                      | Control      | 90 days  | 8.4000     | ±1.07054 | 4.092*       |
|         |                                      | Experimental | 90days   | 8.2000     | ±0.95759 |              |
| 5.      | TC (mg/dl)                           | Control      | 0 day    | 181.9412   | ±6.34341 | -1.354<br>NS |
|         |                                      | Experimental | 0 day    | 183.0000   | ±5.07519 |              |
|         |                                      | Control      | 90 days  | 182.1765   | ±4.35869 | 2.393*       |

|    |              |              |         |          |           |              |
|----|--------------|--------------|---------|----------|-----------|--------------|
|    |              | Experimental | 90days  | 180.6471 | ±4.76020  |              |
| 6. | Tg (mg/dl)   | Control      | 0 day   | 174.0294 | ±11.03022 | 0.606<br>NS  |
|    |              | Experimental | 0 day   | 173.5294 | ±9.84804  |              |
|    |              | Control      | 90 days | 174.7941 | ±10.44112 | 4.725*       |
|    |              | Experimental | 90days  | 171.9118 | ±9.44319  |              |
| 7. | LDL (mg/dl)  | Control      | 0 day   | 109.0471 | ±5.29266  | -1.478<br>NS |
|    |              | Experimental | 0 day   | 110.1176 | ±4.46702  |              |
|    |              | Control      | 90 days | 108.9235 | ±4.55725  | 2.012*       |
|    |              | Experimental | 90days  | 107.5588 | ±4.25855  |              |
| 8. | VLDL (mg/dl) | Control      | 0 day   | 34.8059  | ±2.20604  | 0.606<br>NS  |
|    |              | Experimental | 0 day   | 34.7059  | ±1.96961  |              |
|    |              | Control      | 90 days | 34.9588  | ±2.08822  | 4.725*       |
|    |              | Experimental | 90days  | 34.3824  | ±1.88864  |              |
| 9. | HDL (mg/dl)  | Control      | 0 day   | 38.0882  | ±2.82164  | -0.594<br>NS |
|    |              | Experimental | 0 day   | 38.1765  | ±2.52827  |              |
|    |              | Control      | 90 days | 38.2941  | ±2.26340  | -3.230*      |
|    |              | Experimental | 90days  | 38.7059  | ±2.32938  |              |

<sup>NS</sup> – Not Significant, \* -  $p \leq 0.05$

#### Low Density Lipoprotein

The serum LDL profile of control group of subjects has shown an increased trend. The mean value of LDL in control group of subjects was found to be  $109.0471 \pm 5.29266$  at 0 day. At subsequent observation period of 90 days it was reported to be  $108.9235 \pm 4.55725$  mg/dl. However in experimental group of subjects there was a

decline in the mean LDL values. On 0 day the mean LDL value was  $110.1176 \pm 4.46702$  mg/dl and at the end of 90 days the mean LDL values was  $107.5588 \pm 4.25855$ . Similar significant changes were visible after 90 days (Table 2)

#### High Density Lipoprotein

The quantitative value of HDL in the control group of subjects remains in almost same range throughout the duration of study. It was  $38.0882 \pm 2.82164$  mg/dl at 0 day, and  $38.2941 \pm 2.26340$  at 90 days. In the experimental group of subjects the mean value of HDL at 0 day was  $38.1765 \pm 2.52827$  mg/dl. It risen to  $38.7059 \pm 2.32938$  mg/dl, which was found to be significant when compared to the counterpart mean value of experimental group at 0 day. Both groups were at homogeneity as no significant difference was there at pre stage (Table 2), but after 90 days a significant difference was there in mean values of both groups.

#### Triglyceride

The control group of subjects has shown almost constant level of serum triglyceride during the different follow up periods, the mean values being  $174.0294 \pm 11.03022$  and  $174.7941 \pm 10.44112$ . Contrary to that there was a decline in the triglyceride in experimental group of subjects after 90 days. The mean serum triglyceride level in this group on 0 day was estimated to be  $173.5294 \pm 9.84804$  mg/dl. However, at 90 the mean value was  $171.9118 \pm 9.44319$ .

#### Total Cholesterol

The mean value of total serum cholesterol in control group of subjects was found to be  $181.9412 \pm 6.34341$  mg/dl, where as in experimental group of subjects it was  $183.0000 \pm 5.07519$  mg/dl (Table 1). After 90 days of experimental intervention the total cholesterol in experimental group was reduced to  $180.6471 \pm 4.76020$  mg/dl (Figure 1). This difference was statistically significant. However in control group of subjects no such reduction was noticed (Table 2). Inter group assessment have also shown significant difference in the mean values of Tc of both control and experimental groups.

### **Discussion and Conclusions**

The red blood cells count and hemoglobin concentration are important clinical indicator because they determine the amount of oxygen that blood can carry. Three of the most common measurements are hematocrit, hemoglobin concentration and red blood cells count<sup>11</sup>. The red blood cells are being produced by myeloid tissue i.e. red bone marrow. The factors influencing the process of erythropoiesis i.e. red blood cells production are: (i) hematopoietic growth factors, (ii) some vitamins, and (iii) iron and

copper. Erythropoietin is the most important, most well known hematopoietic growth factor which causes erythropoiesis. Most of the times stimulus for erythropoietin is hypoxia i.e. deficit in oxygen supply. But certain other factors are also involved. It has been reported that high red blood cell count is due to high erythropoietin production<sup>12</sup>. This high red blood cell count helps in bringing in state of homeostasis and enhance the capability of the human subject to battle with the diseases like chronic lung disease and Anemia, which in turn may bring the state of good health and physical well being. Findings of our study have shown increased RBC count and quantitative hemoglobin level following experimental intervention. Such changes are probably maintained through a 'biphasic mechanism – (1) by enhancing the production of normal hematopoietic growth factor thereby enhancing the red blood cell production and (2) by maintaining the normal life span of existing red blood cells. This may be because of the proactive role of perception of psychic center (*Jyotikendra Preksha*) which would have probably influenced the stem cells in Red bone marrow. This hypothesis is in the conformity of the findings reported by Shekhawat<sup>10</sup> and who have elaborated that the RBC production is influenced by certain enzymatic factor related with hematopoietic growth factors erythropoietin.

The erythrocyte sedimentation rate is being used as a screening test for a wide variety of infections, inflammations and certain other pathological states. The test does not distinguish between specific diseases or pathological conditions but somehow it reflects the presence of either type of antigens or pathogens. The tendency of the red blood cells to settle down increases when they form Rouleaux. Rouleaux formation increases when there is increase of plasma fibrinogen  $\gamma$ -globulin. Most infectious antigens cause increase of  $\gamma$ -globulins including fibrinogen. Possibly the yoga and meditation practices influence the  $\gamma$ -globulin level through neurotransmitter and hormonal profile route<sup>11</sup>. Our findings have exhibited a decrease in erythrocyte sedimentation rate following the experimental intervention, which may be taken as the enhancement of the anti-inflammatory immune capacity. The results of this study supports the view that Preksha Meditation modulates the neurotransmitter and hormonal profile through the activation of neurohormonal complex mechanism<sup>1</sup>.

Preksha Meditation causes inhibition of sympathetic nervous system and activation of parasympathetic nervous system<sup>13</sup>, which ultimately decreases the metabolic rate. This may probably be the reason of lower blood glucose level. Similar findings were reported in another research on Preksha Meditation<sup>9</sup>. Maintaining a constant blood glucose concentration seems to be important, particularly since most tissues can shift to utilization of fats and proteins for energy in the absence of glucose. It is so because the glucose is the only nutrient that can be utilized by the brain, retina and germinal epithelium of the gonads in sufficient quantities to supply them with their required

energy<sup>14</sup>. Minimum level of energy is required by brain and retina during the practice of Preksha Meditation because both these organs are remain in dormant phase. This may be the reason that lower blood glucose level might have been reached following the practice of Preksha Meditation.

Quantitative profile of total cholesterol, triglyceride and differential cholesterol have shown a specific trend of change. Total cholesterol, triglyceride, low density lipoprotein (LDL) and very low density lipoprotein (VLDL) were found to be on lower side that too with a statistical significance, whereas high density lipoprotein (HDL) was reported to be increased significantly in the experimental group of subjects practicing Preksha Meditation practice module<sup>9</sup>.

Most of the body's cholesterol is endogenous (internally synthesized) rather than dietary and to some extent the body compensates for variations in dietary intake, high intake somewhat inhibits hepatic cholesterol synthesis. However, the liver synthesizes a certain amount of cholesterol regardless of intake, and severe restriction of dietary cholesterol does not necessarily result in proportionate drawn in blood cholesterol. Dietary fatty acids also strongly influence cholesterol levels<sup>10</sup>. Exercise lowers blood cholesterol levels. The mechanism is somewhat round about, Exercise reduces the sensitivity of the right atrium of the heart to blood pressure, so the heart secretes less atrial natriuretic factor. Consequently, the kidneys excrete less sodium and water, and the blood volume rises. This dilutes the lipoproteins in the blood, and the adiposites compensate by producing more lipoprotein lipase<sup>11</sup>. Thus the adiposites consume more blood triglyceride. This shrink the very low density lipoprotein particles, which shed some of their cholesterol in the process, and high density lipoprotein, pick up this free cholesterol for removal by the liver<sup>15</sup>.

Hormones are the primary regulators of fat metabolism and some important hormones which are involved in this process are insulin, glucagon, epinephrine, norepinephrine, human growth hormone and thyroxin. Probably the most dramatic increase that occurs in fat metabolism is that observed during exercise. This results almost entirely from release of epinephrine & norepinephrine by the adrenal medullae as a result of sympathetic stimulation. Meditation probably modulates the functions of Adrenal gland through hypothalamic- pituitary axis which results in alteration in quantitative release of epinephrine and norepinephrine. Our findings with Preksha Meditation are same lines supporting the hypothesis of alteration in the function of hypothalamus-pituitary combine following yogic practices. Further these two hormones directly activate hormone sensitive triglyceride lipase that is present in abundance in the fat cells and this cause very rapid break down of triglyceride and mobilization of fatty acids<sup>16</sup>. Experimental intervention i.e. Preksha Meditation practice most probably initiates the activation of the parasympathetic nervous system,

resulting in alteration not only in fat mobilizations but also increased ratio of high density lipoprotein to low density lipoprotein.

On the basis of the findings of this study it may be inferred that regular Preksha Meditation practice, even for short duration, helps in bringing the qualitative changes in blood profile which is a detrimental biochemical compound involved in various metabolic processes responsible for the state of physiological health and well being.

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