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## Effect of alterations in breathing patterns on EEG activity in normal human subjects

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### Abstract

Hypoxemia, hyper/hypocapnia due to altered respiratory patterns in patients with chronic respiratory ailments cause changes in cortical functions leading to encephalopathy. But presence of other simultaneous confounding factors in these patients makes it difficult to understand the effect of different breathing patterns alone on cortical areas. Therefore, the present study aim to investigate the effect of different breathing patterns on alpha and beta activity over different brain regions in normal human subjects. To achieve this, thirty healthy male subjects were asked to perform 3-minutes of slow deep breathing (SDB), Breath holding (BH) and fast deep breathing (FDB) while EEG was recorded at frontal, parietal and occipital sites. Percent change in power reveal that both alpha and beta activities increased following FDB in the frontal region (approx 40±10% at F3 & 25±15% at F4) whereas, they decreased in all the regions post BH but only alpha decreased posteriorly post SDB (up to 20±5% at P3 & 18±5% at P4). Therefore, alterations in post-interventional cortical EEG activities seems to have alpha preponderance which might be sensitive to relative hypoxemia/ hypercapnia or hypocapnia, having differential response to the respiratory interventions.

**Keywords:** Slow deep breathing, breath holding, mild hyperventilation, EEG waves.

### Introduction

Respiratory system which is largely autonomic in nature can be transiently modified or modulated by the voluntary control. Such modification if achieved voluntarily (hyper- or hypoventilation or apnea) or occur due to presence of some disease conditions (COPD, Asthma or fibrosis etc), are known to exert influence at different levels of neuraxis including the cortical, autonomic as well as peripheral outputs. In this context, several earlier studies (1, 2, 3, 4) have shown significant

alterations in the sympathetic and parasympathetic responses due to voluntary changes in the respiratory patterns e.g. controlled breathing. It has been reported that apnea by voluntary breath-holding lead to increase in sympathetic activity (5, 6) whereas, voluntary hyperventilation lead to increased sympathetic and decreased parasympathetic response in normal healthy individuals (3). These changes are reflected in terms of alterations in arterial blood

pressure and many other visceral changes along with altered R-R interval in ECG.

Altered respiratory patterns may not only induce hypoxemia, hyper- or hypocapnia leading to changed arterial blood pressure (7) but these hemodynamic changes also cause altered cortical functions (8). EEG findings in COPD patients with encephalopathy/ intellectual impairment show higher occurrence of slow wave bands and lower occurrence of beta waves predominantly in the fronto-temporal region, secondary to hypoxemia & hypercapnia (9,10). Besides, a brief period of hyperventilation is also known to influence CNS by causing generalised increase in excitability helping to reveal latent epileptic foci if any, and hence is used in diagnosis of epilepsy by provoking epileptic discharges (11).

It has been also shown that autonomic features (in terms of changes in heart rate variability indices) and alpha/theta rhythm of EEG show a high degree of correlation during event related attention exercises (12). Studies have also suggested lateralization of cortical management of the autonomic nervous system, with the right hemisphere being principle manager of sympathetic and the left hemisphere of parasympathetic activity (13).

However, very few studies have been done so far to evaluate EEG changes in cortical activity during altered breathing in normal subjects, with maximum findings conducted mainly either during task & stress related events, or on patients with respiratory or neurological diseases. Therefore, in this study we aim to investigate the changes in EEG profile (in terms of alpha and beta waves) across different brain regions during voluntary alterations in the breathing pattern in healthy adult human subjects which might eventually help in understanding the basis of encephalopathy or cognitive impairment seen in COPD and other respiratory disorders patients.

## Materials and Methods

The study was of self controlled prospective experimental type and was conducted on healthy adult male volunteers (n=30) of age group 18-24

years with normal BMI (18.5-24.9). Institutional Ethics Committee clearance was obtained before initiating the study. Informed consent form was taken from each subject and a detailed medical history of all the subjects was also taken to meet the inclusion & exclusion criteria. Any subject with hypertension/ diabetes mellitus/ respiratory diseases /any other disease history which may affect their autonomic function/ history of any neurological or psychological disorder/habits of smoking, alcohol consumption etc. were excluded from the study. None of the subjects were taking any drug / medication for the last two weeks prior to study.

## Protocol for performing altered breathing patterns:

### Slow / Deep Breathing (SDB):

The subjects were asked to breathe deeply and steadily at a rate of 6 breaths per minute (metronome guided) for three minutes (14).

### Breath Holding (BH):

The subjects were made to perform voluntary end-inspiration breath-holding till breaking point (as confirmed by the diaphragmatic flutter in the spirogram) to elicit apnea (15).

### Fast Deep Breathing (Fast-DB):

The subjects were made to breathe deeply and rapidly at a rate of 30 breaths per minute (metronome guided) for three minutes (1).

**Recording of EEG:** EEG was recorded bilaterally from frontal, parietal and occipital regions (F3, F4, P1, P2, and O1& O2) according to the international 10-20 system of electrode placement. Reference electrodes were also placed on the left and right earlobes (A1 & A2) to serve as references. After placement, the impedance of each electrode was checked ( $< 5 \text{ k}$  ). These electrodes were then connected to Digital EEG machine (Recorder & Medicare System, India) through a junction box which contained EEG acquisition and software for Power Spectral analysis of EEG. After digitalization, the raw

signal was submitted to Fast Fourier Transformation (FFT) that computes power for the various EEG frequency bands.

### Experimental Design:

Room temperature of the laboratory was maintained at the thermoneutral zone i.e.  $26 \pm 2$  C. All the recordings were conducted 2-3 hours after the last meal in the afternoon. Heart Rate and Blood Pressure were recorded upon arrival of the subject and again after 5 minutes of rest to ensure that the subject is in resting state before commencing the experiment. Thereafter, EEG was recorded in supine posture with eyes closed and the subject was instructed to be completely relaxed. The resting EEG was recorded till alpha waves were observed for more than 50% of the recording time at the occipital region (16). Then the subjects were asked to perform all the three breathing challenges in the sequence described below. Following each intervention, the subject was allowed to recover till the EEG profile and heart rate (assessed by ECG recording) returned to basal value as observed in pre-intervention condition. At least 10-15 min of resting recording was taken after each intervention. The EEG and ECG recording were continued throughout the procedure.

*Reduction of EEG waveforms:* By visual inspection of EEG records, five artifact free epochs, each of 6 sec, were randomly selected during pre-intervention sessions (17). Similarly, five artifact free epochs of 6 sec each were selected for the first 2 minutes (0-2 min) after each intervention with a gap of 20 sec between each epoch. Thereafter, Fast Fourier Transformation was performed on these data for decomposition of the EEG waveform into sine wave components in terms of respective frequency bands i.e. alpha (8-12 Hz) & beta (15-30 Hz) and power spectral density (PSD in  $\mu\text{V}^2$ )

was estimated for each type. As the inter-subject basal power of EEG waves had large variations among each other, the powers obtained after each post-intervention session for each frequency were expressed as percentage (%) change in relation to respective resting EEG power. The results were depicted in terms of mean of % change  $\pm$  Standard error ( $M \pm SE$ ) for statistical analysis.

### Statistical Analysis:

Two-tailed Mann-Whitney U-nonparametric test was used to compare statistically between absolute power of alpha & beta bands, at frontal, parietal & occipital sites obtained during the pre- and post-intervention periods. A probability of less than 0.05 ( $p < 0.05$ ) was considered as statistically significant.

### Results

All the resting EEG data are being expressed in terms of their standard units, but effects of all the interventions are expressed as % change in relation to their resting values.

(i) **Resting/basal EEG:** All the subjects in supine, relaxed state with eyes closed, had significantly higher alpha power at frontal (F3 & F4), parietal (P3 & P4) and occipital (O1 & O2) sites as compared to beta power. However, there was significantly higher alpha power in parietal and occipital area as compared to frontal site with O1 & O2 having maximum alpha. Overall absolute power of beta had the minimum value during resting state when compared to alpha but had significantly higher power at O1 & O2 compared to its F3 & F4 values (Table 1). All the values were found to be bilaterally symmetrical with no significant inter-hemispheric difference in the respective locations.

**Table 1: Resting absolute power of Alpha & Beta Waves at different cortical sites**

EEG Waves (PSD in $\mu\text{V}^2$ )	Cortical sites of recording					
	F3	F4	P3	P4	O1	O2
<b>Alpha</b>	6.37 $\pm 11.6$	14.2 $\pm 8.39$	72.04 $\pm 45.4^{***}$	64.9 $\pm 56.08^{***}$	106 $\pm 43.4^{***}$	110.64 $\pm 40.9^{***}$
<b>Beta</b>	1.76 $\pm 0.7$	1.78 $\pm 0.8$	2.21 $\pm 0.6$	2.19 $\pm 0.7$	3.55 $\pm 1.2^{***}$	3.24 $\pm 1.1^{***}$

Resting absolute power of cortical alpha and beta waves in left and right frontal, parietal and occipital sites. When compared with frontal areas, significantly high power of alpha at parietal and occipital areas were observed, whereas beta power was significantly more at occipital areas.

\*\*\* $p < 0.001$

**ii) EEG changes after Slow Deep Breathing:**

Within 2 min. of post SDB, there was a significant decrease in alpha activity posteriorly

(P3, P4, O1 & O2). However, no significant changes were observed for beta power in any of the sites (Fig. 1).

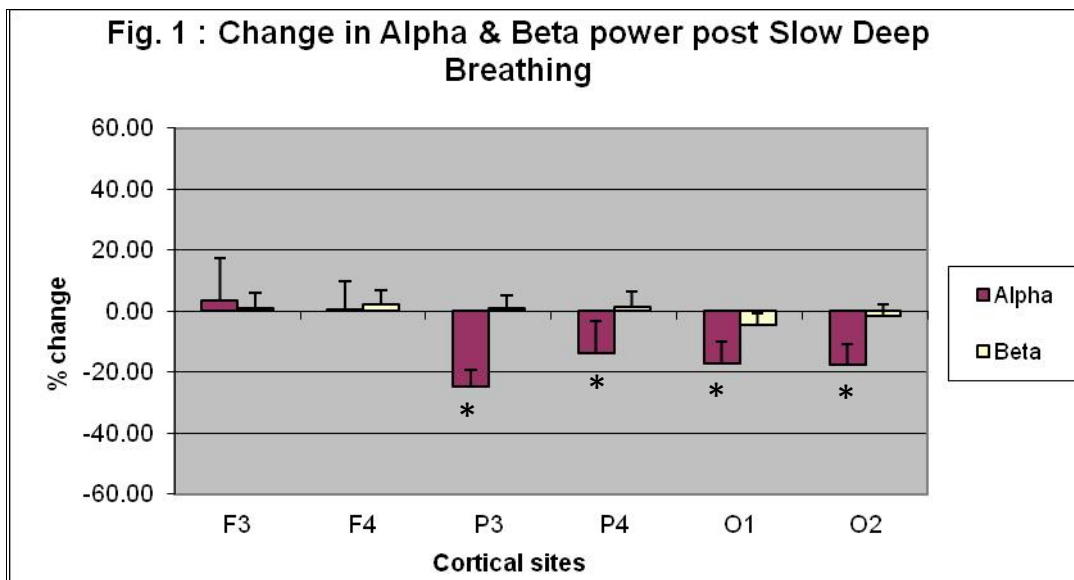


Fig.1: Depicts % change in the power of alpha and beta waves after Slow Deep Breathing at different cortical sites. Significant decrease was observed bilaterally in alpha power at parietal & occipital regions as compared to resting state. No such difference was observed for beta at any of the sites. Values are expressed as Mean $\pm$ SE. \* $p < 0.05$

**iii) EEG changes after Breath Holding:**

Overall, all the EEG waves (alpha & beta) showed decrease in power as compared to resting values following 2 minutes of breath holding (apnea). Out of these, maximal decrease was observed in

alpha activity which was significant at all the recorded cortical sites (F3, F4, P3, P4, O1 & O2). A similar pattern was recorded for beta activity in a smaller magnitude (Fig. 2).

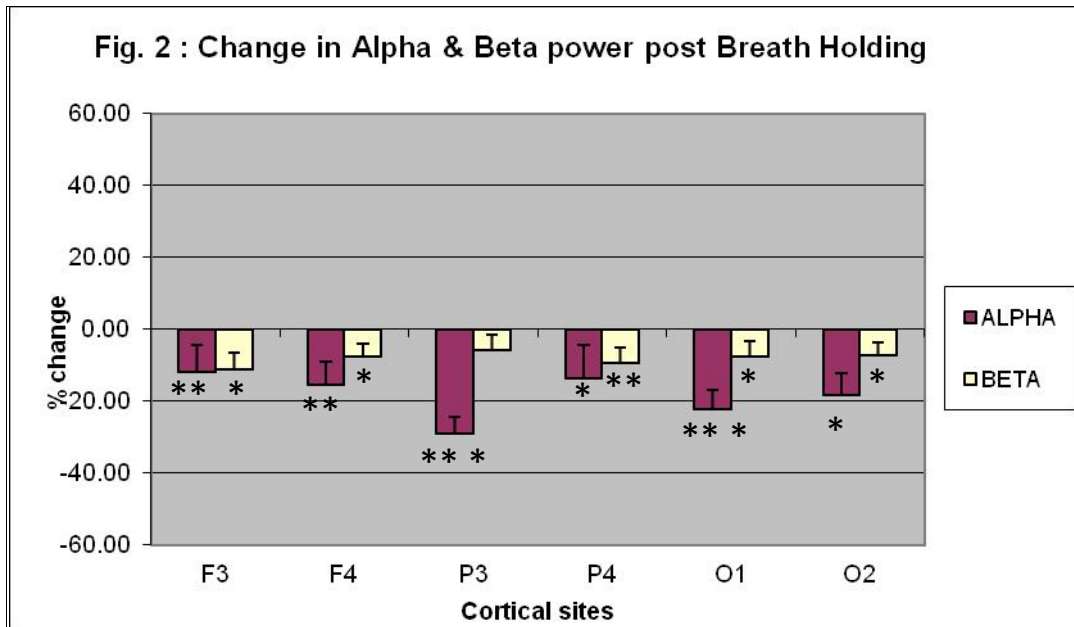


Fig.2: Effect of Breath Holding on the power of alpha and beta waves (% change from resting state) at different cortical sites. Significant decrease was observed bilaterally in both alpha and beta power at all the recorded sites which was more prominent for alpha. Values are expressed as Mean±SE. \*p 0.05, \*\*p 0.01, \*\*\*p 0.001

**iv) EEG changes after Fast Deep Breathing:**

Beta activities were found to be increased at all the recorded sites i.e. F3, F4, P3,P4, O1 & O2,

after 2 min of FDB which were highly significant, whereas, alpha activity showed marked increase at frontal areas (F3 & F4) only (Fig. 3).

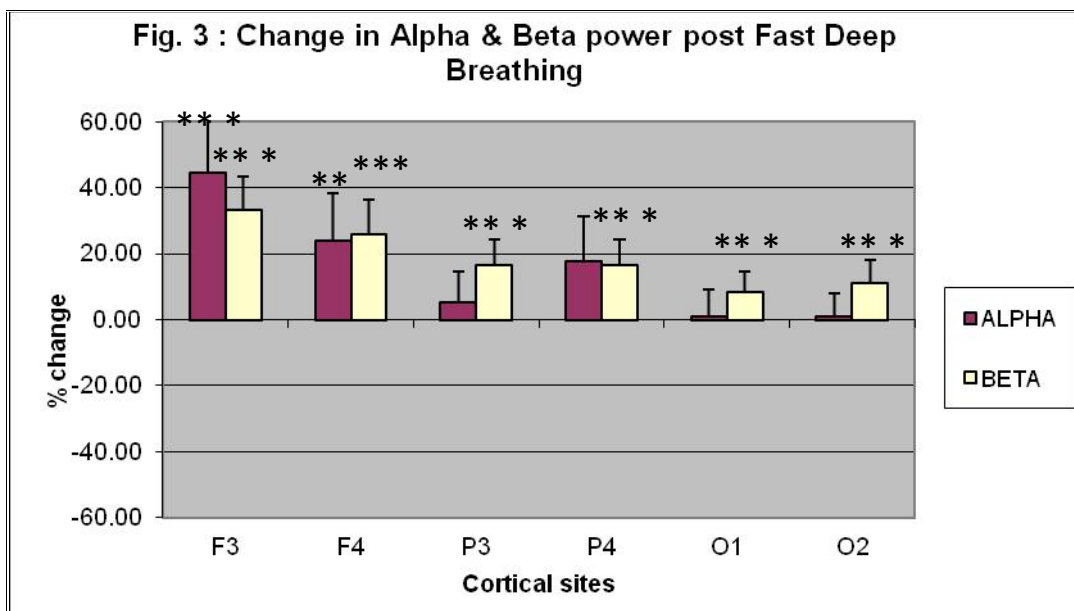


Fig.3: Effect of Fast Deep Breathing on the power of alpha and beta waves (% change from resting state) at different cortical sites. Beta power showed significant increase at all the recorded cortical sites bilaterally whereas alpha showed significant increase only at the frontal sites. Values are expressed as Mean±SE. \*\*p 0.01, \*\*\*p 0.001

## Discussion

In the recent past, various studies were being performed to understand the relation between the cortical activities with the respiratory functioning as both of them are bound to influence each other. Reports are available to show the influence of altered respiratory patterns on cortical neural activity (from EEG & MRI studies) in respiratory patients. In our study, the resting EEG status of the subjects showed predominantly alpha activity in the posterior sites which was highest at the occipital areas. Even at the frontal sites, the alpha power was more than beta power. These findings definitely suggest a true resting state of the subjects (16). The activities showed bilateral symmetry which again corroborate with the standard resting EEG recordings (16).

Decreased alpha power was observed posteriorly following SDB with no significant change in beta activity all over the cortex in our subjects (Fig.1) which may be indicative of generally decrease in response of cortex to deep/slow breathing of short duration (i.e. 3 minutes). This finding is in accordance to the study conducted on Pranayamic breathing (18) where a hypothesis was put forward towards resetting the autonomic nervous system through stretch induced inhibitory signals and hyperpolarization currents caused by voluntary SDB.

Practice of SDB over some time has been found to alter autonomic patterns in different ways, in a few reports (19,20) including bhastrika pranayama. In fact, research has documented clearly that deep breathing induced vagal stimulation is a sensitive measure of parasympathetic cardiac function (21). This is also reported to decrease the stress with increased appearance of relaxed EEG waves i.e. alpha waves (22). However, we did not see any increased alpha which may be due to the fact that it was a one time deep breathing manoeuvre without any prior practice. Besides, subjects were required to follow the audio signal for maintaining a fixed rhythm of 6 breaths/min which requires attention and alertness.

On the other hand, significant decrease in all the recorded waves (alpha & beta) at all the cortical sites starting from frontal to occipital areas was observed following BH. The hypercapnia and hypoxia induced by the BH may be the reason for a general depressed cortical activity observed in this case.

Regarding beta waves, it has been reported that beta waves in the motor cortex are associated with the muscle contractions which are suppressed prior to and during movement changes (23,24) whereas beta activity is increased when movement has to be resisted or voluntarily suppressed (25). This might be true in the present study also when beta waves might have been suppressed immediately following the BH. In this context, it would be interesting to see if an increase of beta activity was present when movement was voluntarily suppressed during BH as may be revealed through future analyses.

While dwelling on the findings post FDB, it may be pointed out here that the protocol of FDB in our study was different (only deep breathing at a rate of 30 per min.) than the classical hyperventilation used during EEG recording to stimulate cortical areas. Classical hyperventilation is more like Maximum Voluntary Ventilation where subjects respire with the deepest breath at the highest possible rate. The mild hyperventilation conducted in our study seem to have more relaxing effect on the cortical activity, thereby causing significant increase in the frontal alpha power in our subjects (Fig 3). At the same time, increased beta power seen all over the cortex post FDB (mild-hyperventilation) may be explained with the fact that during the recording of FDB, the subject was expected to synchronize his breathing rate with the rate given by the experimenter (cycle of inspiration/expiration per 2 secs). This definitely demanded an alert state of the subject which may be the reason for increased beta activity all over. However, looking at the relative power of each wave would be more informative to comment on the effect of FDB. But, for the present study, as we have not included relative power analysis and therefore, we would like to further analyze this data in future study. Therefore, overall, post FDB resulted in

generalized increased state of neural activity but differential response of alpha and beta was observed following SDB.

## Conclusion

From the above discussion it may be concluded that altered respiration induced hypoxemia or hypo/hypercapnia which has direct bearing on the cortical activity causing thereby stimulation or inhibition of different cortical areas. Also, these ventilatory changes in general might not only reflect alterations in cardiac activity of the individuals, but also may be associated with altered pO<sub>2</sub>, pCO<sub>2</sub> and pH leading to hypoxia, hypoxemia, hypercapnia, acidosis and alkalosis, thereby affecting the cortical activity. This also gives the basis of a definite links between cortical activity and autonomic outflow of respiratory system. Further, changes in respiratory patterns among various patients suffering from acute or chronic ventilatory dysfunctions are well known. Thus, the present study suggests a possible link between autonomic and cortical neuronal mechanisms as influenced by altered respiratory patterns in various types of respiratory illnesses.

Further study including analysis of arterial blood gases & respiratory gases on larger sample may provide vital correlates with the observed alterations in neural activity in patients with respiratory compromise.

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