

Brainstem Auditory Evoked Potential Changes in Chronic Obstructive Pulmonary Disease Individuals

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Abstract: COPD is a disease characterized by airflow limitation and is not fully reversible. Cigarette smoke can induce inflammation and directly damage the lungs. It is a major public health problem and currently fourth leading cause of death worldwide. COPD includes emphysema and chronic bronchitis. Auditory brainstem response (ABR) is a click stimulus that generates a response from the hair cells of the cochlea, the signal travels along the auditory pathway from the cochlear nuclear complex to the inferior colliculus in mid brain generates wave I to wave V. Brainstem auditory evoked potentials (BAEP) are the potentials recorded from the ear and vertex in response to a brief auditory stimulation to assess the conduction through auditory pathway up to midbrain. The eighth cranial nerve and brain functions are impaired in COPD patients. BAEP is a valuable tool to evaluate the auditory involvement in COPD patient AIM: To study the BAEP abnormalities in stable COPD patients. OBJECTIVE: To compare the BAEP abnormalities in stable COPD patients with age matched control. Materials and Methods: 30 male COPD subjects who were chronic smokers are recruited from medicine OPD. 30 age matched non smokers healthy volunteers served as the control group from master health check up. BERA recording was done in Neurophysiology Laboratory, Department of Physiology, Stanley Medical College using POLYRITE MEDICAID-Neuroperfect Plus. Parameters taken: latencies of wave I,II,III,IV,V. Statistical analysis done by student independent 't' test, p value < 0.00 considered as significant. Results: the latencies of waves III,IV,V were prolonged in COPD patients. Conclusion: BAEP can be used as a tool to detect earlier the neurological involvement in COPD patients.

Key words: Baep • Copd • Hypoxemia

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a disease state characterized by airflow limitation that is not fully reversible. It is a major public health problem and presently fourth leading cause of death worldwide. COPD is presently regarded as a multi-system disorder. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.

Pathogenesis of COPD includes imbalance of proteinases and anti proteinases in the lung and the oxidative stress [1]. Cigarette smoke can also induce inflammation and directly damage the lungs. COPD is one of the major causes of chronic respiratory failure. As the disease advances, hypoxemia develops as a result of ventilation/perfusion imbalance, decreased diffusion

capacity and alveolar hypoventilation [2]. Hypoxemia increases particularly in the exacerbation periods of the disease. When arterial partial oxygen pressure (PaO₂) falls below 60 mmHg tissue hypoxia occurs and this causes systemic effects. Visual and auditory receptors are affected from hypoxia [3, 4].

COPD includes emphysema and chronic bronchitis. Emphysema is destruction and enlargement of lung alveoli. Chronic bronchitis is characterized by chronic cough and sputum. Pathological changes in the lungs includes mucus hypersecretion, ciliary dysfunction, airflow limitation, pulmonary hyperinflation, gas exchange abnormalities, pulmonary hypertension and cor pulmonale. They usually develop in this order over the course of the disease. Hereditary deficiency of alpha-1 antitrypsin, asthma and airway hyperresponsiveness, lung growth are the host factors.

Tobacco smoking, Indoor air pollution (such as biomass fuel used for cooking and heating) Outdoor air pollution, Occupational dusts and chemicals are the main risk factors. Smoking, long-lasting hypoxia and age have been the contributing factors for the development of peripheral neuropathy in patients with chronic pulmonary disease. The diagnosis of COPD was based on modified criteria defined in the Global initiative for chronic Obstructive Lung Disease (GOLD) guidelines [5].

Brainstem Evoked Response Audiometry (BERA) are the potentials recorded from the ear and vertex in response to a brief auditory stimulation to assess the conduction through auditory pathway up to midbrain. Auditory brainstem response (ABR) is a click stimulus that generates a response from the hair cells of the cochlea, the signal travels along the auditory pathway from the cochlear nuclear complex to the inferior colliculus in mid brain generates wave I to wave V. BERA has expanded the possibility of objective testing of conduction in auditory pathway and hearing functions. It is an effective and simple method requires less cooperation of the subjects, measures the intactness of specific part of the auditory pathway. In this study we aimed to evaluate the changes in BAEP in COPD patients.

Objective: To compare the Brainstem Auditory Evoked Potential in chronic obstructive pulmonary disease individuals with age matched controls.

MATERIALS AND METHODS

Sample Size: 30 The present study has been conducted in the department of neurophysiology lab, Stanley Medical College, Chennai. Ethical approval from the Institutional Ethical Committee, Stanley Medical College, Chennai was obtained for the study.

Study Group: 30 COPD subjects who were smokers from Stanley Medical College Hospital

Control Group: 30 age and gender matched healthy non smokers from the Master Health Check Up.

The patients who had a stable course of disease with regular follow-up in 1 year and no hospitalization for COPD-related illness during the preceding 6 months were included in the study.

Inclusion Criteria: COPD with /without asthma Chronic Smokers (10-15 cigarettes /day for >5yrs) Males of

30-40yrs, Duration of illness in all patients with COPD was more than 5 years.

Exclusion Criteria: Carcinoma lung, Females of that age, other respiratory infection, Intake of ototoxic drug, Hearing loss, psychological or neurological problems, chronic alcoholism, uremia, cystic fibrosis, sarcoidosis, leprosy, malignancy, any hereditary disorders involving peripheral nerves, history of intake of any neurotoxic drug, Lesion occupying intracranial space, Neuromuscular junction disorder, Diabetes, Hypertension, coronary artery disease.

The participants were made to relax and be comfortable prior to the tests. A detailed clinical history about COPD was collected. Written and Informed consent obtained before subjects entered the study. Pure tone audiometry done prior to the recording. Complete external ear examination was done for both ears. Wax removed. The basic parameters of subjects height weight, age were recorded.

BERA recording was done in Neurophysiology Laboratory of Research Wing, Department of Physiology, Stanley Medical College using Polyrite Medicaid-Neuroperfect Plus. The left and right ear tested separately in all subjects. The laboratory temperature was maintained uniformly. Recordings were done in the forenoon 10.00 am to 12 p.m. in sitting posture after a light breakfast.

Subjects were informed to take shampoo bath of scalp and strictly oil free on the day of recording. Disc electrodes placed on scalp by 10-20 standard system with conducting jelly. This provided stability of electrodes and prevented infection. The reference electrode was placed on the vertex (Cz) and active electrode on the side of mastoid on which side of ear stimulated. Ground electrode connected on the right forearm (Fz). Electrode impedance was checked.

Amplifier was on since the biological signals are very small. Automatic artifact rejection was used. Sweep velocity was 1 millisecond. Click acoustic stimuli at a rate of 11 pulse per second at an intensity of 90dB hearing level to the ear stimulated and masking sound of 40dB in non stimulated ear was given through head phone supplied by Medicaid. Electrical activity had low cut filters Hertz and high cut filters 10Hertz in order to avoid any electrostatic and electromagnetic interferences including the domestic switches. 1000 auditory click responses were summated and averaged and displayed. Rarefaction was chosen in which, ear head phone diaphragm moves away during auditory clicks [6].

Table 1: latencies of the left ear

Wave-latency (ms)	Control, n=30 mean±STD	COPD n=30 mean±STD	P-value
I	1.72±0.057	1.82±0.154	0.22
II	2.43±0.164	2.45±0.069	0.30
III	3.7±0.08	4.41±0.30	0.00
IV	5.15±0.07	5.33±0.31	0.00
V	5.85±0.11	6.13±0.23	0.00

p value<0.00is highly significant.Statistical analysis by student independent 't' test.

Table 2: Latencies Of The Right Ear

Wave -Latency(ms)	Control n=30,mean±STD	COPD, n=30mean±STD	P-value
I	1.80±0.09	1.86±0.11	0.04
II	2.71±0.07	2.74±0.23	0.48
III	3.72±0.10	3.74±0.08	0.38
IV	5.10±0.08	5.3±0.29	0.00
V	5.72±0.11	6.8±0.23	0.00

p value< 0.00 is significant, Statistical analysis by student independent 't' test.

RESULTS

Over left ear, the latencies of waves III, IV and V in COPD patients were prolonged significantly as compared to the healthy volunteers.

Over the right side, there was significant prolongation of the latencies of waves IV and V in COPD group as compared to the healthy volunteers.

The mean and standard deviations for each variable were calculated in control group and COPD group separately. The statistical significance between two groups was analyzed by using independent sampltestt and p value <.00 was considered statistically significant.

DISCUSSION

In our study, COPD patients with mild-to-moderate airflow obstruction and with no clinical features of neuropathy were included. Our objective was to assess the impaired brainstem auditory evoked potentials instable COPD patients.In this study, all COPD patients were significant smokers and clinically presented with airflow limitation.

Smoking affects the ponto-medullary portion of the brain. The contents of cigarette smoke leads to hypoxia and BAEP abnormalities. Nicotine and carbon monoxide may actually deplete oxygen levels to the cochlea [7]. A predominant feature of hypoxia is that it slows information processing. Increase in BERA latency of COPD is more significant for the later waves (III, IV, V) than for the earlier waves. The results suggest that there is central nervous system involvement in addition to

peripheral auditory impairment. The common BAEP abnormalities observed in COPD patients in my study include prolongation of latencies of waves III, IV V.

Kayacan *et al.* [8] observed that smoking, airways obstruction and long-lasting COPD may not only cause peripheral neuropathy but may also affect the ponto-medullary portion of the brain due to hypoxemia, hypercapnia and respiratory acidosis. Atis and co-workers studied BAEP in patients with severe COPD and concluded that eighth cranial nerve and brainstem functions were impaired in COPD [9]. It has been hypothesized that the abnormal BAEP findings are due to brainstem hypoxia which increases with the severity of COPD. In my study as COPD patients happened to be heavy smokers, the possibility of the contents of cigarette smoke will be the cause leading to BAEP abnormalities.

CONCLUSION

The present investigation concluded that BAEP abnormalities can be used as a tool to assess the auditory changes in for COPD patients.

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