

Effect of Passive smoking on bronchial reactivity in asthmatic patients**Hussain S Al-Janabi****College of Medicine , Babylon University , Department of Medicine****Abstract:**

The effect of passive cigarette smoke exposure on the bronchial reactivity of 10 asthmatic patients was studied and compared with (10) apparently healthy people as control. Measurements of vital capacity (VF) and forced expiratory volume in one minute (FEV₁) and pulmonary flow-volume curve (PF, FEF₇₅, FEF₅₀, FEF₂₅) were obtained before and after 30 minutes exposure to side-stream smoke in a simulated tobacco combustion chamber. Post exposure values were significantly reduced ($p < 0.05$) in all variables which were more pronounced in asthmatic patient compared with control. FVC and FEV₁ reduced by 22.5% and 27.9% respectively. Air flow in mid (FEF₅₀) and end (FEF₂₅) airways was reduced by 32.5% and 45.8 % receptively such findings clearly demonstrated the serious effect an the ventillatory function of the asthmatic patients exposed to a side-stream smoke in a relatively closed environment.

الخلاصة:-

يهدف البحث إلى معرفة وقياس الاثار الضارة للتدخين السلبي على مرضى الربو . تم قياس مؤشرات وظائف الرئة لعشرة مرضى وعشرة أشخاص للسيطرة . لوحظ تدهور وظائف الرئة عند مرض الربو بعد تعرضهم لدخان السجائر في غرفة مغلقة وانخفضت مؤشرات القياس بنسب ملحوظة وبدلالات عضوية حيث انخفضت كفاءة الرئة وحجم الزفير في الدقيقة الأولى بنسب 22.5% و 27.9% على التوالي . كذلك تم قياس سريان الهواء في منتصف ونهايات القصبات حيث انخفضت بنسب ذات دلالة معنوية وهي 32.5% و 45.8% على التوالي .

Introduction:

Inhaled tobacco smoke can produce an acute increase in airway resistance and passive exposure to tobacco smoke correlates with respiratory symptoms such as cough, wheeze and sputum production (1).

The non specific hyperirritability of bronchial tree that underlying pathogenesis of asthma may well be triggered by irritants contained in the cigarette smoke. Although allergens can trigger episodes of asthma in atopic patients but asthma is more often aggravated by nonspecific factors such as cold air , tobacco smoke, dust and acrid fumes (2). Passive smoking has been reported to effect pulmonary function in asthmatic patients. (3,4,5,6). Airway limitation and more serious impairment of lung function has been caused by passive exposure to cigarette smoke (4,7,8,9) . Cigarette smoke has it's main effect on the peripheral airflow limitation (2,10,11) passive smoking is also reported to be associated with increasing incidence of asthma (9,11) . Tobacco smoke is known to interact and adds to the effects of all other factors affecting broncho-pulmonary function (FVC, FEV₁, and PF, FEF₂₅₋₇₅). The aim of this study is define the magnitude of derangement in

airflow measurements in asthmatic patients passively exposed to cigarette smoke in a relatively closed environment like being a partner to a smoker person, a member of smoking family or a workmate to group of smoker labourers.

Subject and Methods:

1. Patient Population:

TEN patient with bronchial reactivity i.e currently or previously asthmatics, were recruited. Diagnosis based on clinical history, Examination, data from previous records and pulmonary functions tests. Ten apparently healthy people, nonsmoker were included as control group.

2. Study Design

- 2.1. The study patients if they were smoking are requested to abstain at least for an overnight and during the day of assessment.
 - 2.2. All medications including bronchodilators, were not allowed at least the day of experiment. Patients who were too ill to stop treatment were excluded.
 - 2.3. The lung function room in Al-Najef Teaching Hospital, 3x4 m, with tightly closed windows was used as tobacco combustion chamber simulate in which both patients and controls were seated. This is a simulation to any tobacco smoke polluted, relatively closed and ill-ventilated environment.
 - 2.4. A health volunteer was asked to smoke cigarettes of an average of 10-15 cigarettes during a chosen period of 30 minutes to provide adequate exposure to side-stream smoke without risking patients (4).
- #### **3. Lung function test**
- were done before and immediately after 30 minutes exposure for both groups (patients and control subjects). The variables measured are parameters of airflow functions i.e forced vital capacity (FVC), forced expired volume in the first minute. (FEV₁) and forced expiratory flow between 25-75 percent of vital capacity (FEF 25-75%).
- #### **4. Data analysis :**
- Done by analysis of variance significance (paired tests). level of significance $P < 0.05$.

Results:

The following table displays measurements of airflow variables studied as parameters for pulmonary response to 30 minutes exposure to aside-stream smoke. FVC, FEV₁ and FEF25-75% were measured before and after exposure for both groups (control and asthmatic). The calculated percent reduction in post exposure values was significant ($P < 0.05-0.005$) for nearly all parameters and in both groups but was more so in asthmatic patents, in comparison with control subjects.

Mean Data of pulmonary response to 30 minute passive smoke exposure in both groups

	Before Exposure Mean+SD	After Exposure Mean+SD	%Reduc.	T.test
FVC				
Control gr.	4.82(+.346)	4.422(+.496)	8.33	p.<0.05
Asthmatic gr.	3.298(+.310)	2.556(+.362)	22.5	p.<0.005
FEV₁				
Control gr.	3.865(+.669)	2.827(+.561)	26.9	p.<0.005
Asthmatic gr.	3.005(+.252)	2.166(+.373)	27.9	p.<0.005
FEF 75%				
Control gr.	8.342(±.273)	7.315(.836)	12.3	p.<0.1
Asthmatic gr.	5.763(±.727)	3.929(±.767)	31.8	p.<0.05
FEF 50%				
Control gr.	5.812(±.282)	4.209(±1.05)	27.5	p.<0.005
Asthmatic gr.	4.586(±.227)	3.11(±.513)	32.1	p.<0.005
FEF 25%				
Control gr.	3.12(±.384)	2.289(±.706)	26.6	p.<0.05
Asthmatic gr.	2.59(±.409)	1.403(±.387)	45.8	p.<0.005

Discussion:

It is long known that people with bronchial hyper reactivity like Asthmatic patients can have definite airflow obstruction with experiencing symptoms of shortness of breath (12). Asthmatic patients characteristically displays bronchial reactivity and hyper responsiveness to a wide range of stimuli (13). Active smoking has a definitive effect on bronchial reactivity and smoking one cigarette was shown to increase the airway resistance significantly and immediately (14).

Previous reports have also demonstrated that normal people can produce significant-increases in airway resistance when exposed to side-stream cigarette smoke (7). Asthmatic patients are reported to be adversely affected by passive smoking (3, 4, 6). By measuring daily peak flow rate, investigators have shown that environmental exposure to tobacco smoke does adversely affect both normal and asthmatic people (5). However, many studies including our work have shown that asthmatic patients are more severely affected than their normal counter parts. The more sever effect on asthmatic patient has been attributed to the larger than normal intrapulmonary deposition of inhaled environmental tobacco smoke particles (15). It seems that particles contained in the cigarette smoke would deposit in an already hyper reactive bronchial walls triggering hypersensitive bronchial receptors leading to an increase in the airway resistance. Such explanation is supported by our study. We have found that the acute exposure to side-stream cigarette smoke for 30 minutes has produced a significant increases in air way resistance in all parts of bronchial tree i.e from proximal to end air ways. Airflow variables FEF75, FEF50 and FEF25 have significantly decreased by 31.8%, 32.1% and 45.8% respectively (shown in the table). A previous investigator (Dahm 1981) had reported a quantitatively lesser response to 30 minutes exposure to side-stream smoke in expiratory flow rates compared to our study. He achieved a decrease of 19.2% and 21.4% in FVC and FEV₁, respectively compared to percent reduction of 22.5% and 27.9 in both FVC , FEV₁ in this study. The differences might be attributed to different methodology, study design and different psychological and emotional responses of this different populations.

Conclusion and Recommendation:

The findings of this work clearly demonstrated the detrimental effects of passive tobacco smoke on bronchial reactivity and pulmonary function particularly of the asthmatic patients.

We conclude that patients with bronchial hypersensitivity should be a ware and exercise extreme caution with regard to not only active cigarette smoking but equally so to a side stream tobacco smoke at home, work place or public facilities to avoid worsening of lung function and to lessen morbidity. Moreover, and taken in consideration, the enormous burden of cigarette smoke on health, we recommend that health authorities and nongovernmental organizations should endorse an active sustained and well organized nation- wide campaign against smoking .

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