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Research article

A COMPARATIVE STUDY OF ARTERIAL STIFFNESS INDICES BETWEEN SMOKERS & NON SMOKERS

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ABSTRACT

Arterial stiffening is recognized as a critical precursor of cardiovascular disease. Smoking is one of the modifiable risk factor for cardiovascular disease. Lifestyle modification is clinical efficacious therapeutic interventions for preventing and treating arterial stiffening. Hence, the current study is designed to compare the Arterial Stiffness Indices between smokers & non smokers. The study involved 55 non smokers & 55 smokers within the age group of 30-50 years. Peripheral Pulse Wave was recorded by Digital Volume Pulse in both non smokers & smokers. Arterial stiffness indices were calculated. **Arterial Stiffness Index = Patients Height / Transit time** [Transit time → Time delay between systolic peak & Diastolic peak] **Reflection Index = Magnitude of Diastolic peak / Magnitude of Systolic Peak.** Arterial Stiffness Index & Reflection Index were highly significant in smokers than nonsmokers, $p<0.001$. The increased arterial stiffness indices in smokers suggest that the cigarette smoke damages the vascular endothelium.

Keywords: Arterial stiffness, Cardiovascular diseases, Digital Volume Pulse, Smoking.

INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of mortality and morbidity worldwide.[1] Arterial stiffness and wave reflection exerts adverse effects on cardiovascular function. Arterial stiffening is recognized as a critical precursor of CVD and is independent predictors of cardiovascular events. Arterial stiffening is a major factor in cardiovascular disease because of the reduced elasticity in blood vessels.[2] Therefore, assessment of arterial stiffness is believed to be useful in the prevention of cardiovascular disease. Smoking is one of the modifiable risk factor for CVD.[3] Tobacco, especially cigarette smoking is a major cause of CVD, responsible for about one third of CVD deaths. Chronic cigarette smoking has been shown to be associated with increased arterial stiffness.[4] The risk of CVD deaths increases with increasing exposure to cigarette smoke, as measured by number of cigarette smoked daily, the duration of smoking & the degree of inhalation & the age of initiation. The relative risk for CVD is substantially greater in early adult life than in old age & is associated more strongly with the cigarette smoke than other forms of tobacco. The pathophysiological

changes in smoking are due to changes in the vascular endothelium, induction of coronary vasoconstriction and changes in the basal nitric oxide (NO) or endothelial nitric oxide synthase production. Lifestyle modification, aerobic exercise and sodium restriction in diet appears to be clinical efficacious therapeutic interventions for preventing and treating arterial stiffening.

Arterial stiffness can be measured using invasive and non-invasive methods. Pulse wave analysis is one of the methods used to assess arterial stiffness. The most popular non-invasive methods are based on pletysmographic principles.[5] A Pletysmographic measurement depends on Boyle's law, a principle that describes the relationship between the pressure & volume of gas. Pulse wave velocity (PWV) is known as an established index of arterial stiffness. But it is difficult, time consuming and expensive. These factors have fostered the development of simple methods to record arterial stiffness. These techniques are much simpler, non-invasive, economical and easier to apply. We can also measure Arterial stiffness Indices from Digital Volume Pulse (DVP) which is economical, easier, less time consuming, non-invasive method. So the present study is designed to record Arterial Stiffness Index (SI), Reflection Index (RI) by using Finger Photoplethysmography & to compare it with smokers and nonsmokers.

MATERIALS & METHODS

The study was conducted in a sample of one ten subjects in Salem. They had been divided into fifty five non smokers (control group) & fifty five smokers (study group) within the age group of 30-50 years. Subjects were selected based on inclusion and exclusion criteria.

Inclusion criteria: Fifty five normal healthy nonsmokers between 30 and 50 years were included in the control group, Fifty five smokers between the ages 30 and 50 years were included in test group.

Exclusion criteria : History of Hypertension, Diabetes Mellitus, Cardio vascular disease, Peripheral vascular disease, Other drug treatment

Methodology: The subjects were selected by a detailed history & thorough physical examination. They were asked to fill a questionnaire. To assess their smoking habits. The experimental protocol was fully explained to the participants to allay apprehension. They were refrained from smoking for 12 hours before the test. Informed consent was taken from all the subjects. The study was approved by the Institutional Ethical Committee.

Experimental design: Data was collected by recording the DVP. Subject's weight was measured using a calibrated weighing machine in light clothing and bare feet. Height was measured in meters. All experiments were performed at room temperature. Baseline pulse rate, Systolic, Diastolic & pulse pressure was measured in sitting position after 5 min of rest by using mercury sphygmomanometer.

Finger Photoplethysmography:

Digital Volume Pulse was measured by an instrument known as Finger Photoplethysmography, using Infra-red light with wave length of 940 nm; placed on the right index finger of the subject. The signal from the instrument was digitalized by digital converter with a frequency of 100 Hz; which was connected to the computer. The main principle of this device is conversion of pressure changes to voltage changes by means of the pressure transducer. Subjects were initially acquainted with the instrument and a trial is given before performing the study. DVP recording was done with the help of software virtual oscilloscope which was provided by national instrument which can be freely distributed for academic purpose. Pulse wave contour consists of two main components: the first component is caused by systolic pressure wave that results from blood ejected from the left cardiac chamber to aorta and its consequent distribution to peripheral sites.

The second component is formed by pressure wave reflected back to the aorta.

Analysis: From the DVP recordings, we estimated arterial stiffness index (SI) & reflection index (RI). The shape of the pulse wave is determined by a number of factors, age, sex, body height, pulse and physical fitness.[6] Length of this travelling wave is usually proportional to the subject's height (h). The time delay between systolic peak & diastolic peak is called Pulse transit time (PTT or ΔT), is inversely proportional to arterial stiffness. The time difference between the peaks of the two components, known as the reflection time, is inversely proportional to arterial stiffness. To correct for the size of the subject, the reflection time is divided by the height of the subject.[5] The resultant value is SI, which is expressed in meters/second. SI is comparable to the definitive measure of arterial stiffness, the pulse wave velocity (PWV). RI is a measure of vascular tone. It is calculated by dividing the amplitude of the systolic component by the amplitude of the diastolic component. This ratio is expressed as a percentage. The parameters and their definitions are shown in Figure 1. These parameters were measured by software Image tool.

Statistical analysis: The results were expressed as mean ± standard deviation (SD). A p value of <0.05 was considered statistically significant. Statistical analysis was performed using the statistical package for social & sciences. Student unpaired't' test was applied to compare between the parameters.

RESULTS

Fifty five normal healthy nonsmokers in the age group of 30 and 50 (36.85 ± 4.99) years and Fifty five smokers in the age group of 30 and 50 (37.29 ± 4.76) years were subjected to DVP recording. Arterial stiffness was estimated from the pulse wave analysis. Both SI (11.74 ± 4.12) meters/second & RI (75.64 ± 12.35) % in smokers were significantly higher at 95% confidence interval than non-smokers SI (5.72 ± 0.28) meters/second & RI (48.19 ± 9.51) %, p < 0.001. The results are shown in the table 1.

Table 1: Comparison of stiffness indices between Smokers & nonsmokers*

Parameters	Non Smokers	Smokers	'p' value
Stiffness index (m/s)	5.72 ± 0.28	11.74 ± 4.12	< 0.001
Reflection index (%)	48.19 ± 9.51	75.64 ± 12.35)	< 0.001

*Data presented as mean ±SD

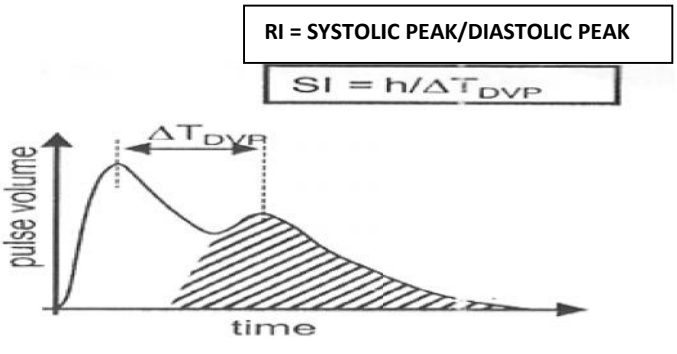

Fig:1. Pulse wave contour and definitions of evaluated parameters

Arterial Stiffness Index (SI) = Patients Height (h)/ Transit time (ΔT_{DVP})

[Transit time (ΔT_{DVP}) → Time delay between systolic peak & Diastolic peak]

Reflection Index (RI) = Magnitude of Diastolic peak / Magnitude of Systolic Peak × 100

DISCUSSION

Measuring arterial stiffness provides good data on the endothelial condition. Smoking is known to increase arterial stiffness in adults.[7-9] Cigarette smoke enhances the atherosclerotic changes by several mechanisms. Endothelial damage is a central feature in the evolution of vascular disease induced by cigarette smoking and may act as a precursor for future atherosclerosis. The major health effects of cigarette smoke include: cancer, noncancerous lung diseases; atherosclerotic diseases of the heart and blood vessels; and toxic to the human reproductive system. Despite the damaging effects of tobacco use; quitting smoking has immediate and long term health effects such as improved circulation and fall in heart rate.[10] The inhalation of cigarette smoke results in activation adrenergic mechanism as the nicotine contained in tobacco stimulates the sympathetic nerve terminals, with consequent systemic release of adrenaline and nor adrenaline. The released catecholamine's bind to α1-adrenergic receptors on vascular smooth muscle to cause muscle contraction and thus a reduction in arterial distensibility and vasoconstriction.[8,11] In the healthy adult, who does not usually smoke, the vasoconstrictor response is soon counterbalanced by the local release of vasodilators from endothelium. The two best characterized endothelial vasodilators are nitric oxide (NO) and prostacyclin. Nitric oxide is considered to be the dominant local regulator of resting vasomotor tone, with packets of NO being produced at regular intervals.[12] Impaired nitric oxide production and endothelial dysfunction have also been known to play major roles in altering the mechanical properties of large arteries.[8, 11]

Smoking cessation is an important lifestyle measure for the prevention of cardiovascular disease, and patients with myocardial infarction may experience as much as a 50% reduction in risk of re-infarction, sudden cardiac death.[13] Quitting in late in life also has positive effects. The toxins from cigarette smoke can go everywhere as the blood flows. Chronic tobacco smoking is associated with endothelial dysfunction. Smoking not only accelerates endothelial dysfunction in the large arteries but it is also responsible for changes in the physical properties of arterioles and small arteries.[14] Vascular endothelium produces a number of mediators including nitric oxide (NO) which regulates arterial wall stiffness. McWeigh et al showed that cigarette smoking triggers NO production damage. Basic structural factors determining arterial stiffness are predominantly collagen, elastin and transmural pressure.[15] The mechanisms involved in amelioration in arterial stiffness with smoking cessation may include lipid-soluble smoke particles,[16] endothelial dysfunction,[17] or vascular inflammation,[18] because smoking cessation leads to reduction in levels of inflammatory markers.[19] Although it may take more than a decade to reverse these vascular changes, and the effect is relatively small, smoking cessation helps to reduce cardiovascular events through amelioration in arterial stiffening.

Willett et al reported that cigarette smoking affects cholesterol metabolism, it lowers levels of the protective high-density lipoprotein (HDL) cholesterol [20] and Rabkin et al reported smoking cessation raises HDL cholesterol.[21] In animal models, nicotine can damage the inner lining of blood vessels, thus enhancing the transfer of low-density lipoprotein cholesterol particles across the arterial wall and into the developing cholesterol-laden plaque.[22] Cigarette smoking also can affect the blood clotting system, including adherence of blood platelets to the lining of arterial blood vessels[23] and the formation of blood clots that block a narrowed artery. Selley et al reported that Acrolein in cigarette smoke is partly responsible for its

platelet-adhering effects.[24] Cigarette smoke also causes spasm of the coronary arteries.[25] Many chemical components of cigarette smoke have been found to accelerate the development of atherosclerotic disease. Nicotine, the major psychoactive component of smoke, causes powerful changes in heart rate and blood circulation. Nicotine appears to cause injury to the arterial lining.[22] Smoking causes tissue injury induced by oxidative stress. Free radicals in cigarette smoke, which are highly reactive oxygen products, are damaging to the heart muscle cells. Studies have reported dermal applications of cigarette smoke in laboratory animals demonstrated chemicals in cigarette smoke underwent covalent binding with heart tissue DNA.[26] Van Schooten et al reported that cigarette smokers showed that the heart tissue contained more DNA adducts than that from nonsmokers and linear relationship between DNA adduct levels and daily cigarette smoking.[27] Furthermore, higher DNA adduct levels were associated with a higher degree of coronary artery disease.

In our study we used SI which substitutes pulse wave velocity (PWV). It has been proved that SI positively correlates with PWV.[28] SI values are mainly influenced by large artery stiffness but it can be also supported by wave reflection from peripheral sites as well as from large arteries (28, 29). For smokers we detected significantly higher values of SI than nonsmokers, $p < 0.001$, which indicates increased arterial stiffness. To determine vascular tone we used the parameter RI. RI was also significantly higher in smokers compared to nonsmokers, $p < 0.001$, indicating vascular tone is increased.

CONCLUSION

There is pronounced increase in SI and RI indicating increased arterial stiffness. This suggests that even young smokers have damaged vascular endothelium. Arterial stiffness determined by Digital Volume Pulse is simpler, less time consuming, economical, easier to apply and non-invasive method. Non invasive measurements of arterial stiffness will aid the optimal stratification of CVD risk in an apparently healthy population. Chronic smoking is a leading risk factor in development cardiovascular diseases. DVP can be used to assess how chronic smoking impairs arterial elasticity by evaluating SI and RI. Since the scientific evidences are suggestive of smoking induction of endothelial dysfunctions, the best advice to the smokers is “stop smoking”.

Scope for the study: Additional study including a detailed evaluation of endothelial factors & measurement of arterial stiffness by Pulse wave velocity is needed to clarify whether initial changes of cardiac impairment exist with early initiation of smoking.

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