THE EFFECTS OF AN EXERCISE PERIOD ON THE OXYGEN SATURATION LEVELS OF SMOKERS AND NON-SMOKERS

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

One of the most fundamental problems facing man is an adequate supply of oxygen. It behooves him to study and solve any problem that may hamper an adequate amount of this life-maintaining element. With the advance of industrialization and a growth of population, many problems have risen that complicates man's access to oxygen. Air pollution and stripping of the plant growth are examples of these problems. Ricci[1] gives the composition of the atmospheric air as 20.93% oxygen, 0.03% carbon dioxide, and 79.04% nitrogen (including trace gases). The additions of foreign substances and irritants and the breakdown of the carbon dioxide-oxygen cycle of plants will alter the percentages of the atmosphere to the detriment of man as shown in a study by Holland and Reid[2]. The Public Health Service Review:1967[3] shows many cases of illness with a direct relationship to air pollution such as heart disease, emphysema, sinusitis, and lung cancer.

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[1] Benjamin Ricci, Physiological Basis of Human Performance, (Philadelphia, 1967), p. 11.

[2] W.W. Holland and D.D. Reid, "The Urban Factor in Chronic Bronchitis," Lancet, February 27, 1965, pp. 445-448.

[3] United States Public Health Service, The Health Consequences of Smoking: A Public Health Service Review:1967, U.S. Department of Health Education and Welfare, Public Health Service Publication No. 1696, (Washington, 1968), pp. 108-110.

It remained for man to bring upon himself one of the detriments to his supply of oxygen. This being the use of tobacco. There no longer remains any doubt that the use of tobacco in its many forms, especially smoking, is harmful to man. The Surgeon General's report of 1964[4] concluded: "Cigarette smoking is a health hazard of sufficient importance in the United States to warrant remedial action." Many individual studies also illustrate the harmful effects of tobacco use. Nodel and Comroe[5], in a well controlled study demonstrated that fifteen puffs of cigarette smoke in 5 minutes caused an average decrease in airway conductance of 34% in 36 normal subjects. Research studies of the harmful effects of tobacco use are noted as far back as a study by W.A. Hammond[6] in 1856.

Respiratory and circulatory functions are affected adversely by smoking. In order to understand fully these effects of tobacco on his fundamental oxygen supply man must examine minutely the cardio-respiratory processes. Respiration according to Ricci[7] is a mechanical-chemical-neural process which involves the delivery of oxygen via the blood stream to the cell in exchange for the end-products of cell

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[4]United States Public Health Service, Smoking and Health. Report of the Advisory Committee to the Surgeon General of the Public Health Service, U.S. Department of Health, Education and Welfare, Public Health Service Publication No. 1103, (Washington, 1964), p. 387.

[5]J.A. Nodel and J.H. Comroe, Jr., "Acute Effects of Inhalation of Cigarette Smoke on Airway Conductance," Journal of Applied Physiology, XVI (1961), pp. 713-716.

[6]W.A. Hammond, "The Physiological Effects of Alcohol and Tobacco Upon the Human System," American Journal of Medical Science, XXXII (1856), pp. 305-320.

[7]Ricci, p. 110.

metabolism. The gaseous phase of cigarette smoke contains about four per cent carbon dioxide. This quantity can increase the levels of carboxyhemoglobin saturation of cigarette smokers from 2% to 10%. Diehl[8] states the potentially harmful gases in cigarette smoke include carbon monoxide in a concentration four hundred times that considered a safe level in industrial exposure. Carbon monoxide combines with hemoglobin in red blood cells, thereby reducing the oxygen-carrying capacity of the blood. In the blood of an average smoker two to six per cent of the hemoglobin is inactivated by carbon monoxide and in a heavy smoker up to 8%. This concentration of carbon monoxide will remain in the blood of a habitual smoker in varying amounts even overnight up to thirty per cent. Since carbon monoxide has a greater affinity for hemoglobin than does oxygen, it literally drives oxygen from the blood. Exposure for one hour to a concentration of carbon monoxide of 120 parts per million causes inactivation of about 5% of the body's hemoglobin and commonly leads to dizziness, headache, and lassitude. Concentrations of carbon monoxide as high as one hundred parts per million often occur in garages, in tunnels, and behind automobiles. Such concentrations are tiny in comparison with those (42,000) parts per million) found in cigarette smoke. This reduction of oxygen decreases one's ability to perform strenous activity and makes smokers "short of breath" upon exertion. This is emphasized by Dr. Harry Gibbons, Chief of Aero-Medical Research, Civil Aero-Medical Institute, Federal Aviation Agency,[9] "A person who smokes a pack of

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[8] Harold S. Diehl, Tobacco and Your Health: The Smoking Controversy (New York, 1969) p. 41.

[9] The Daily Oklahoman, June 4, 1969, p. 4.

cigarettes a day is physiologically 7,000 feet in the air while he's on the ground."

DeVries[10] in explaining heart function stated that the function of the heart and the circulatory system is to provide the flow of blood necessary to maintain homeostasis of the various tissues of the body. Homeostatis can be defined as the sum total of regulatory functions that maintain a constant environment for the cells of the tissue.

Purpose of Study

The purpose of the study was threefold: (1) To find if there is a difference in the resting oxygen saturation level between smokers and non-smokers. (2) To determine if there is any difference in the effects of exercise on the oxygen saturation levels between smokers and non-smokers. (3) To determine if a relationship exists between oxygen saturation levels during and after exercise and work load as indicated by pulse rate.

Definitions

The term oxygen saturation was used in this study as the proportion of the hemoglobin which is combined with oxygen and is expressed as per cent of saturation. Blood contains 0.25 volumes per cent dissolved oxygen so 99% of the oxygen transported must be carried in combination with hemoglobin and not in physical solution. For the

[10]Herbert A. DeVries, Physiology of Exercise for Physical Education and Athletes, (Dubuque, 1966), p. 60.

purpose of this study a smoker was defined as an individual that used at least twelve cigarettes per day. A non-smoker was defined as an individual that did not smoke to any degree.

Hypothesis

The Null Hypothesis was used for the purposes of this study. This hypothesis was that there were no significant differences between the resting pulse rates and oxygen saturation levels of smokers and non-smokers. Further, that there was no significant difference between the oxygen saturation levels of smokers and non-smokers during an exercise period.

Limitations

A random sample was not used in conducting this study as the subjects were volunteers from physical education classes and fellow workers of the writer. A majority of the subjects tested were athletes and physical education majors; therefore, the results would not necessarily reflect the oxygen saturation levels and pulse rates of the adult male population. The size of the groups was comparatively small and would be a limiting factor in applying the results to the adult male population.

Assumptions

It was assumed that valid measures were obtained from the measuring devices and that the subjects were free from any heart or blood circulation problems that would have prevented accurate readings.

CHAPTER II

REVIEW OF RELATED LITERATURE

Many studies have been made on the subject of oxygen since its discovery by Harvey. Because of its importance to life there is little that has not been reported on its actions and functions. Likewise, the studies of the effects of tobacco, though much more recent than that of oxygen, cover many volumes of research, discussion, and results of studies made to inform of the results of tobacco use. Although many of these studies involve the effects of tobacco use on the oxygen metabolism of the body few of them can be related specifically to the purpose of this study.

Parkinson and Koefod[1] studied the effect of cigarette smoking on the breathlessness of exertion (descending and climbing once at a quick walking pace a flight of 25 stairs) in 10 healthy soldiers and 20 cases of "soldiers heart" (effort syndrome), all of whom were smokers. The authors concluded that the smoking of 4 to 5 cigarettes could render healthy men more breathless on exertion, and manifestly did so in a large portion of cases of "soldiers heart."

In a study at Antioch College in 1925, Earp[2] examined 172 non-smokers, 35 moderate smokers, and 70 heavy smokers for: (a) standing

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[1] J. Parkinson and H. Koefod, "The Immediate Effect of Cigarette Smoking on Health Men and On Cases of "Soldiers Heart." Lancet, II, 1917, pp. 232-236.

[2] J.R. Earp, "Tobacco, Health, and Efficiency," Lancet, I, 1925, p. 213.

pulse rate; (b) reclining pulse rate; (c) difference between a and b; (d) pulse rate after exercise; (e) difference between d and a; (f) systolic blood pressure standing; (g) systolic blood pressure after exercise; (h) difference between g and f; (i) diastolic blood pressure standing; (j) difference between f and i. As measured by these tests, it was said to be obvious that smoking, in the degree carried on by Antioch students, did not materially affect the functional efficiency of the cardiovascular system.

Reindell and Winterer[3] studied the circulatory effects of cigarette smoking on 14 smokers and 16 non-smokers following 50 knee bend exercises. In most individuals, smokers as well as non-smokers, the pulse rate was increased, and blood pressure variations of ±30 mm. Hg occurred immediately after exercise, the fluctuations in blood pressure making an average figure give a false picture. Schlipp[4] tested 10 smokers and 10 non-smokers for the effect of smoking one cigarette on heart rate during and following an exercise consisting of 36 one-foot step-ups per minute for 2 minutes; each subject performed the exercise 3 times without having smoked previously, and 3 times just after smoking, and a continuous record of the heart rate during and after exercise was made. Although both sitting and standing pulse rates were significantly higher after smoking, at no time was the heart rate during exercise after smoking significantly different from that when no

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[3] H. Reindell and R. Winterer, "Untersuch Ungservnisse Uberdie Wirkung des Rauchens auf Den Kreislauf," Zschr. Klin. Med. CXLI, 1942, pp. 228-287.

[4] R.W. Schlipp, "A Mathematical Description of the Heart Rate Curve of Response to Exercise with Some Observations on the Effects of Smoking," Research Quarterly, XXII, 1951, pp. 439-445.

smoking preceded the exercise. In other words, although on smoking days, the subjects went into the exercise period with higher heart rates, this quickly adjusted itself so that absolute heart rates, not just the increases, coincided during the exercise period on both smoking and control days.

The development of the oximeter as discussed by Millikan[5] gave an insight into the oxygen carrying capacity and saturation levels not before possible. Many researchers in the physiology of exercise field such as Karpovich,[6] DeVries,[7] Ricci,[8] Margaria,[9] and Morehouse,[10] have worked with this instrument and others to present the many facets of what occurs in the cardiovascular functions of the body during exercise.

Shepard[11] discussed cardiovascular functions in regard to oxygen saturation. He says,

> that at any given value for diffusing capacity the oxygen saturation of the blood leaving the alveolar capillaries

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[5]G.A. Millikan, "The Oximeter, An Instrument for Measuring Continuously the Oxygen Saturation of Arterial Blood in Man," Review of Scientific Instruments, XIII, (May, 1942), pp. 434-444.

[6]Peter V. Karpovich, Physiology of Exercise, (6th Ed., Philadelphia, 1965), p. 55.

[7]DeVries, p. 86.

[8]Ricci, p. 117.

[9]R. Margaria, Principia di Chimica e Fisico Chimica Fisiologica, (8th Ed., Milano, 1958), p. 168).

[10]Laurence E. Morehouse and Augustus T. Miller, Physiology of Exercise, (St. Louis, 1948), p. 63.

[11]R.H. Shepard, et al., "Factors Affecting the Pulmonary Dead Space as Indicated by Single Breath Analysis," Journal of Applied Physiology, XI, 1957, pp. 241-243.

> is very little affected by increasing oxygen consumption until a critical point is reached. When the oxygen consumption increases beyond this point, the oxygen saturation of the blood drops precipitously. Direct measurements of the oxygen saturation of the arterial blood show that the normal person exercising at sea level cannot increase his oxygen consumption enough to cause unsaturation of the arterial blood.

The effects of smoking on oxygen saturation were stressed by McFarland[12] in reporting that a value of only 5 per cent carboxyhemoglobin saturation, not uncommon in smokers, creates a physiological state of anoxia equivalent to being at an elevation of 8,000 feet, with an arterial oxygen saturation of only 91 per cent. This was emphasized by the work of Richards[13] on the effects of diffusion factors of mountain climbers. It is now well recognized that the diffusion gradient at the end of the alveolar capillary is much larger at low levels of ambient oxygen tension than at sea level and that at low ambient oxygen tension the diffusing capacity has a much more important effect upon the arterial oxygen saturation. Calculations indicate that a large diffusing capacity is needed to prevent extreme hypoxemia when the oxygen uptake is increased at high altitude. To the extent that hypoxia resulting from inadequate diffusing capacity limits the ability to perform physical exercise at high altitude, it may be said that exercise tolerance is "diffusion limited."

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[12] R.A. McFarland and A.L. Mosely, "Carbon Monoxide in Trucks and Buses and Information from Other Areas of Research on Carbon Monoxide, Altitude, and Cigarette Smoking," Conference Proceedings: Health, Medical, and Drug Factors in Highway Safety, National Academy of Sciences, National Research Council Publication No. 328, (Washington, 1954), pp. 417-433.

[13] D.W. Richards, Jr., "The Lewis A. Connor Memorial Lecture: The Nature of Cardiac and Of Pumonary Dysphea," Circulation, May 1, 1953, pp. 7-15.

Since cigarette smokers have a chronically elevated carboxyhemoglobin level, usually greater than 2 per cent and occasionally exceeding 10 per cent,[14] a study by Chevalier[15] was performed having non-smokers inhale enough carbon monoxide to raise their carboxyhemoglobin levels to the range seen in a control group of cigarette smokers. This maneuver caused the development, in the study group of non-smokers, of an increased oxygen debt with exercise and a reduced pulmonary diffusing capacity at rest. These changes after carbon monoxide inhalation were similar to those found without carbon monoxide inhalation when comparing cigarette smokers to non-smokers.

A series of experiments were done by Krumholz and his associates[16, 17, 18] to evaluate cardiopulmonary function in young apparently healthy persons. The first experiment[19] involved 18 house staff physicians ranging in age from 24 to 37 years. Nine had smoked at least one pack of cigarettes a day for the preceding 5 years and

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[14]McFarland, p. 421.

[15]R.B. Chevalier, R.A. Krumholz, and J.C. Ross, "Reaction of Non-Smokers to Carbon Monoxide Inhalation," Journal of the American Medical Association, CXCVIII, (1966), pp. 1061-1064.

[16]R.A. Krumholz, R.B. Chevalier, and J.C. Ross, "Cardio-pulmonary Function in Young Smokers," Annals of Internal Medicine, LX, (1964), pp. 603-610.

[17]R.A. Krumholz, R.B. Chevalier, and J.C. Ross, "Changes in Cardio-pulmonary Functions related to Abstience from Smoking," Annals of Internal Medicine, LXII, (1965) pp. 197-207.

[18]R.A. Krumholz, R.B. Chevalier, and J.C. Ross, "A Comparison of Pulmonary Compliance in Young Smokers and Non-Smokers," American Review of Respiratory Diseases, XCII, (1965), pp. 102-107.

[19]Krumholz, Annals of Internal Medicine, LX, 603-610 ff.

nine had not smoked for at least the same time period. Extensive pulmonary function studies were done at rest and after exercise. The smokers were found to have a greater oxygen debt after exercise, decreased diffusing capacity at rest and with exercise, and decreased total lung capacity and vital capacity.

In the second Kromholz experiment[20] 10 young staff physicians, all of whom had smoked at least one pack a day for five years, were given pulmonary function tests immediately after and again 3 weeks after abstinence from smoking. Six physicians refrained from smoking for 6 weeks and were tested again. After 6 weeks of no smoking, expiratory peak flow and pulmonary diffusing capacity were significantly increased. Heart rate and oxygen debt after exercise were decreased. After 6 weeks functional residual capacity was decreased and inspiratory reserve volume and maximal voluntary ventilation were increased.

The final Krumholz study[21] again used 20 young medical persons divided among 10 smokers and 10 non-smokers. The mean pulmonary compliance was significantly greater for the non-smokers than for the smokers.

A study of the immediate effects of smoking was conducted by Fodor, Glass and Weiner[22] on 400 students at San Fernando Valley

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[20]Krumholz, Annals of Internal Medicine, LXII, 197-207 ff.

[21]Krumholz, American Review of Respiratory Diseases, XCII, 102-107 ff.

[22]J.T. Fodor, L.H. Glass, and J.M. Weiner, "Immediate Effects of Smoking on Healthy Young Men," Public Health Reports, LXXXIV, (1969), pp. 122-127.

State College. As part of the study, work-performance efficiency level was compared between a smoking group and a non-smoking group. The results showed an increase in the smoker's pulse rates during the smoking period, the work period, and the resting period. In addition, because of a higher ratio of oxygen uptake and carbon dioxide release the work task required more effort by the smokers.

Summary

The effects of tobacco use on the body have been studied for over a century. There have been many varied techniques and results as were shown in the literature. Early studies that compared smokers and non-smokers such as those by Earp,[23] Reindell and Winterer,[24] and Schlipp[25] were concerned mainly with pulse rate and blood pressure increases. The findings of these studies did not seem to show that smoking had any effect on the cardiovascular system.

The more refined nature of the research of the men in physiology of exercise helped establish the effects of smoking during exercise on the cardiorespiratory systems. Although the findings of Shepard[26] and McFarland[27] seem to be contradictory, the effects of the gaseous stage of smoking, especially that of carbon monoxide, as emphasized by

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[23]Earp, p. 213.

[24]Reindell and Winterer, pp. 228-287.

[25]Schlipp, pp. 439-445.

[26]Shepard, pp. 241-243.

[27]McFarland, pp. 417-443.

Chevalier,[28] undoubtedly create abnormal conditions such as the hypoxia resulting from inadequate diffusing as noted by Richards.[29]

The Krumholz[30, 31, 32] experiments seem to point out very distinctly the deteriorating effects of smoking on the cardio-pulmonary functions.

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[28] Chevalier, pp. 1061-1064.

[29] Richards, pp. 7-15.

[30] Krumholz, Annals of Internal Medicine, LX, 603-610 ff.

[31] Krumholz, Annals of Internal Medicine, LXII, 197-207 ff.

[32] Krumholz, American Review of Internal Medicine, XCII, 102-107 ff.

CHAPTER III

METHODOLOGY

This study was conducted in the Physiology of Exercise Laboratory, Colvin Physical Education Center on the Oklahoma State University Campus during the spring of 1969. The tests were given between 9 AM and 4 PM. The laboratory was air conditioned with constant temperature of approximately 72^{0} F.

Subjects

The subjects were thirty-two adult males ranging from age 19 to age 44. Thirteen of the subjects were undergraduate physical education majors, 8 were varsity athletes, 5 were physical education graduate students, 3 were graduate students from other departments, 2 were air force enlisted personnel, and 1 was a member of the physical education faculty. Fifteen of the subjects were smokers and 17 were non-smokers.

The smoking group had smoked for an average of 9.6 years. The range of daily cigarette consumption was a low of 12 to a high of 35. The average daily cigarette consumption was 22.3 per day.

Measuring Devices

Pulse rate measurements were taken with the aid of an E and M Instrument Company Telemetry System consisting of a transmitter, Model FM-1100-E2, and a Bio-Telemetry Receiver, Model F-M-1100-7.

The receiver was connected to an E and M Instrument Company Physiograph for recording purposes. The recording paper was set at a speed of 2 cm per second. The paper was marked at 1 sec. intervals to assist in counting pulse rate. The oxygen saturation level was obtained through a Waters Company XP-350 Oximeter. The oximeter was attached to the subjects by means of an earpiece which contains a photo-electric cell that refracts light beams passing through the ear and is sensitive to changes in oxygen concentration. The oximeter apparatus includes a sphygmomanometer to pressurize the earpiece during certain oximeter settings. The exercise portion of the testing was done on a Quinton Treadmill set at a standard speed of three and one-half mph and a standard grade of 12%. This represents a rise of twelve feet for every 100 feet traveled on a horizontal plane.

Experimental Design and Procedure

Telemetry electrodes were attached to each subject, one over the lower sternum and the other electrode approximately three inches to the left and 3 inches below the left nipple. The telemetry receiver and the physiograph were adjusted and the resting, standing pulse rate of the subject was recorded. The subject was stationed on the treadmill and the earpiece of the oximeter was attached to the pinna of the ear. The standing, resting oxygen saturation level was then obtained and recorded.

The pulse rate was again checked for proper recording. When all instruments were working properly the subject was instructed on the treadmill procedure. The treadmill was started and a ten minute walking exercise period was started. At the end of each one minute interval the oxygen saturation level was observed and recorded. A periodic check of the pulse rate recording was made to insure an accurate and legible count.

To check the reliability of the instruments and procedure a retest was run on two smokers and on 2 non-smokers.

The records for the resting pulse rate, the resting oxygen saturation level, the exercise pulse rate, and the exercise oxygen saturation level were totaled, averaged, and comparisons made between smokers and non-smokers. t-ratios were used to test for significance of differences between means where means were compared. The five per cent level of confidence was established as the criterion of significant difference. To determine the relationship between work-load and oxygen saturation level, a correlation was made between peak pulse rate and the lowest oxygen saturation level for each subject. Another correlation to indicate the same relationship was made between the group mean pulse rate and the group mean oxygen saturation level for each minute of exercise.

CHAPTER IV

RESULTS

The results of the experiment were plotted graphically for resting and exercise period readings. The results for the smoking group are shown in graph 1 and the results for the non-smoking group are shown in graph 2.

Resting Pulse Rate

The group classified as smokers had a mean resting pulse rate of 74.93 beats per minute. The non-smokers had a mean resting pulse rate of 66.82 beats per minute. The difference between the means of the groups produced a t-ratio of 3.650. A t-ratio of 2.038 is required for significance at the 5 per cent level of confidence with 32 degrees of freedom. Therefore the difference between the means of the groups was statistically significant.

TABLE I

MEAN RESTING PULSE RATES

	Smokers	Non-smokers	t-ratio
Mean Resting Pulse Rate	77.93	66.82	3.650*
Standard Deviation	5.554	6.581	

*This t-ratio was significant at the 1 per cent level of confidence

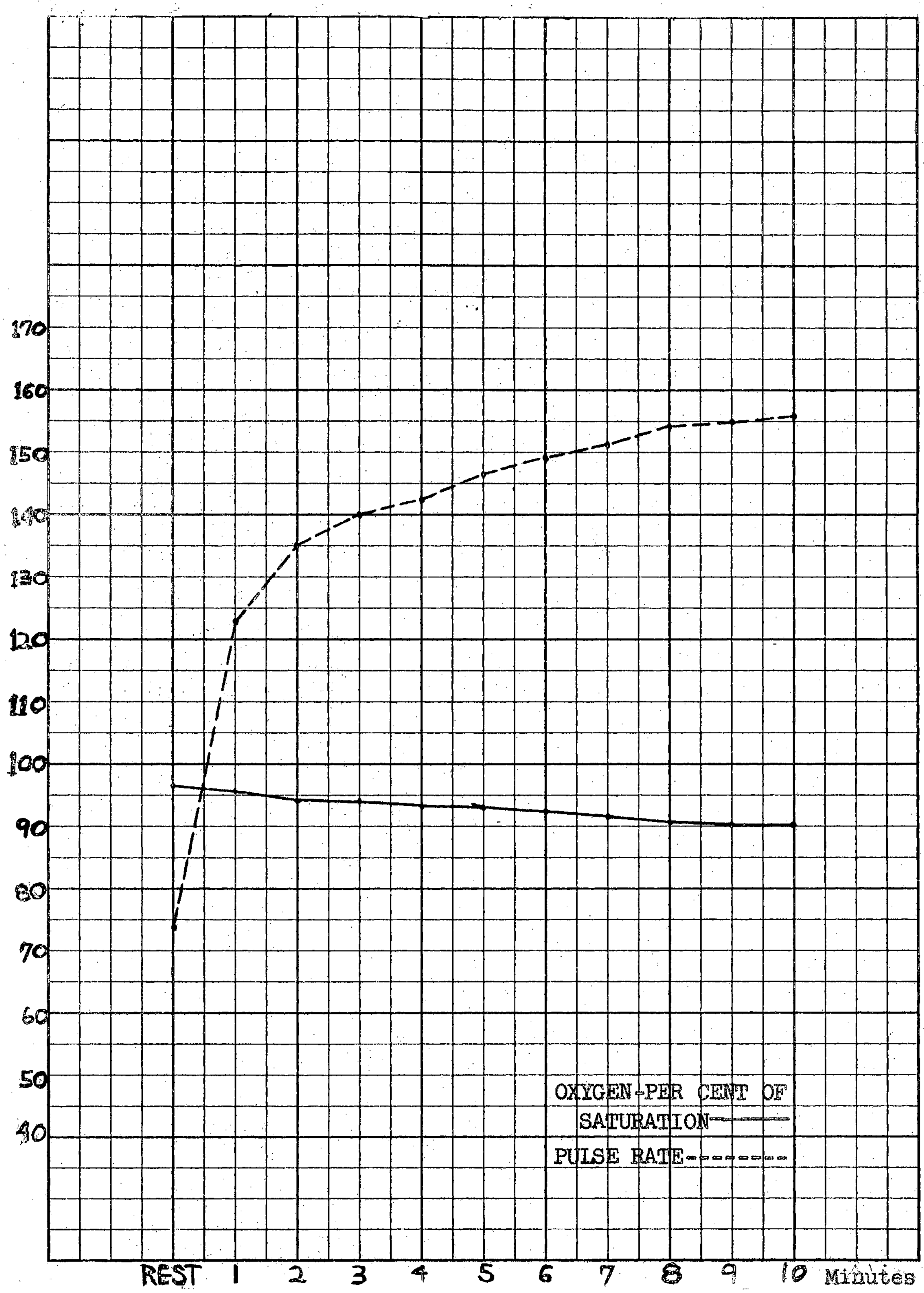

Figure 1. Smokers Pulse Rates and Oxygen Saturation Levels at Rest and During Exercise

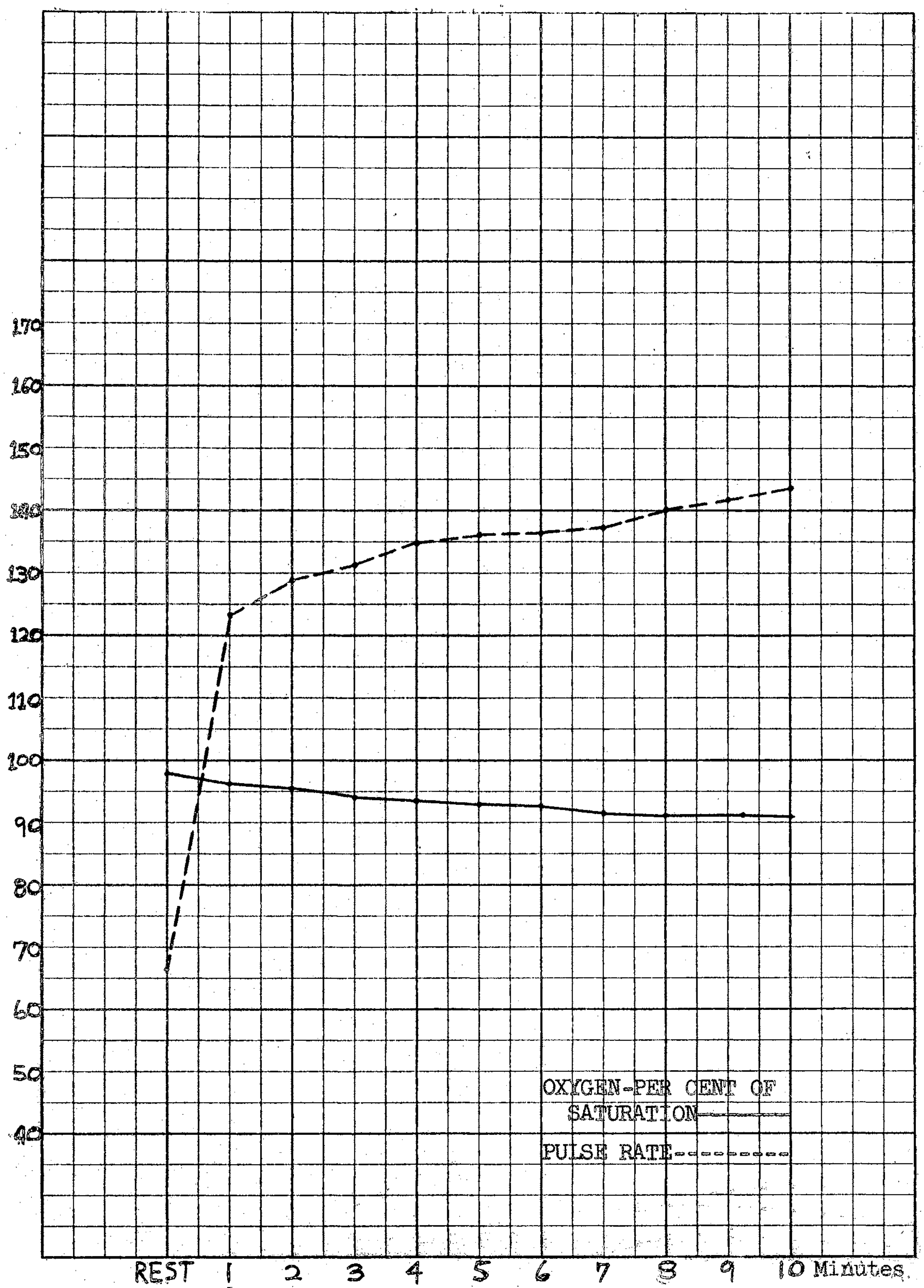

Figure 2. Non-Smokers Pulse Rate and Oxygen Saturation Levels at Rest and During Exercise

Resting Oxygen Saturation Level

The smoking subjects had a mean resting oxygen saturation level of 96.44 per cent. The non-smoking group had a mean resting oxygen saturation level of 97.33 per cent. The difference between the means of the groups produced a t-ratio of 3.102. A t-ratio of 2.038 is required for significance at the 5 per cent level of confidence with 32 degrees of freedom. Therefore, the difference between the means of the groups was statistically significant.

TABLE II

MEAN RESTING OXYGEN SATURATION LEVELS

	Smokers	Non-smokers	t-ratio
Mean Resting Oxygen Saturation Level	96.44	97.33	3.102*
Standard Deviation	.436	1.048	

*This t-ratio was significant at the 1 per cent level of confidence

Exercise Pulse Rate

The smokers group had a mean exercise pulse rate of 156.80 beats per minute at the end of the ten minute exercise period. The non-smokers group had a mean exercise pulse rate of 142.82 beats per minute at the end of the ten minute exercise period. The difference between the means of the groups produced a t-ratio of 2.541. A t-ratio of 2.038 is required for significance at the 5 per cent level of confidence with 32 degrees of freedom. Therefore, the difference between the means of the groups was statistically significant

TABLE III

MEAN EXERCISE PULSE RATES

	Smokers	Non-smokers	t-ratio
Mean Exercise Pulse Rate	156.80	142.82	2.541*
Standard Deviation	13.30	16.79	

*This t-ratio was significant at the 1 per cent level of confidence

Exercise Oxygen Saturation Level

The smoking group had a mean exercise oxygen saturation level of 90.07 per cent at the end of the ten minute exercise period. The non-smoking group had a mean exercise oxygen saturation level of 91.63 per cent at the end of the ten minute exercise period. The difference between the means of the groups produced a t-ratio of 4.831. A t-ratio of 2.038 is required for significance at the 5 per cent level of confidence with 32 degrees of freedom. Therefore, the difference between the means of the groups was statistically significant.

TABLE IV

EXERCISE OXYGEN SATURATION LEVELS

	Smokers	Non-smokers	t-ratio
Mean Exercise Oxygen Saturation Level	90.07	91.63	4.831*
Standard Deviation	0.721	1.034	

*This t-ratio was significant at the 1 per cent level of confidence

Oxygen Saturation Levels

The oxygen saturation level mean at rest for the entire group of smokers and non-smokers was 96.50 per cent. The oxygen saturation level mean at the end of the ten minute exercise period for the entire group of smokers and non-smokers was 90.85. The difference between the scores resulted in a t-ratio of 6.257. A t-ratio of 2.038 is required for significance at the 5 per cent level of confidence. Therefore, the difference between the means of the oxygen saturation levels was statistically significant for two measures of the same group.

TABLE V

MEAN RESTING AND EXERCISE OXYGEN SATURATION LEVELS

	Resting	End of Exercise	t-ratio
Total Group Oxygen Saturation Level	96.50	90.85	6.257*

*This t-ratio was significant at the 1 per cent level of confidence

Correlations

A correlation was calculated between the scores of the lowest oxygen saturation level and the highest pulse rate of each subject. This correlation was -0.4583. A correlation of 0.339 is required to be significant at the 5 per cent level of confidence with 32 degrees of freedom. Therefore, the correlation between the lowest oxygen saturation levels and the highest pulse rates was statistically significant at the 1 per cent level of confidence.

Another correlation was calculated between the group means of the oxygen saturation levels and the pulse rates at the end of each minute of the ten minute exercise period. This correlation was -0.6153. A correlation of 0.576 is required to be significant at the 5 per cent level of confidence with 10 degrees of freedom. A correlation of 0.708 is required to be significant at the 1 per cent level of confidence with 10 degrees of freedom. Therefore, the correlation between the oxygen saturation levels and pulse rates at the end of each minute of the ten minute exercise period was significant at the 5 per cent level of confidence but was not significant at the 1 per cent level of confidence.

Changes in Pulse Rate and Oxygen Saturation Levels

The increase in pulse rate during the ten minute exercise period is shown in graph 3. There was 8.9 beats per minute difference in the resting pulse rate between smokers and non-smokers. At the end of the first minute of exercise the pulse rate for both groups had increased to approximately the same level of 123 beats per minute. From this point the non-smokers pulse rate did not increase as rapidly as the smokers pulse rate. Both groups had a slow but steady rise in pulse rate from the first exercise minute through the tenth exercise minute. At the end of the tenth minute of exercise the smokers had a group mean pulse rate of 156.80 beats per minute while the non-smokers had a mean group pulse rate of 142.82 beats per minute. This was a difference of 13.98 beats per minute between the groups at the end of the exercise period.

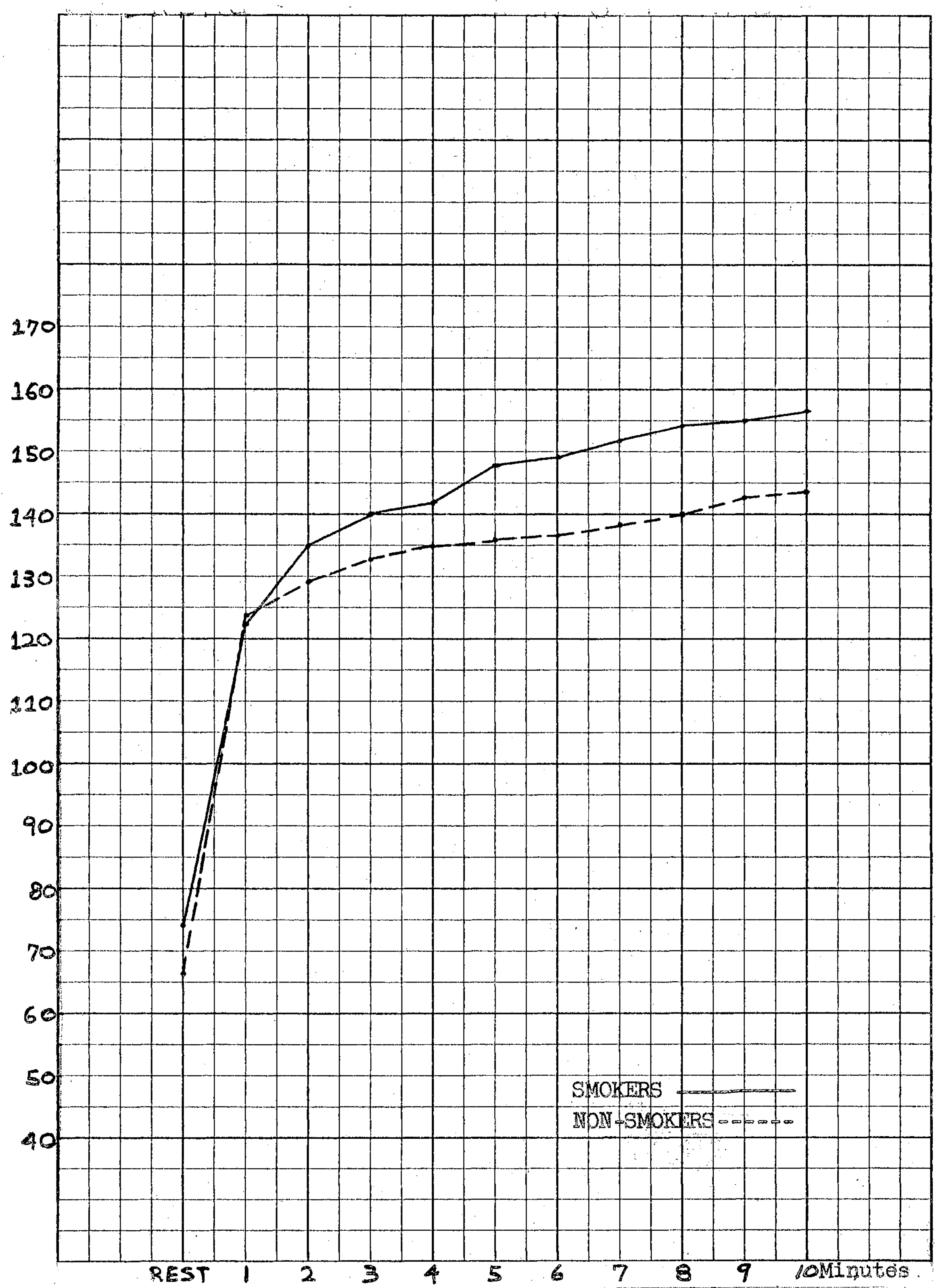

Figure 3. Group Pulse Rate Level at Rest and During Exercise

Graph 4 shows the changes in the oxygen saturation levels of the groups during the exercise period. The resting oxygen saturation level group mean for smokers was 96.44 per cent. The resting oxygen saturation level group mean for non-smokers was 97.34 per cent. The difference between the groups was 0.90 per cent. Both groups had a steady decrease of the oxygen saturation level during the exercise period except the smoking group had a leveling off period between the second and third minutes. The difference in the oxygen saturation level of the groups at the end of the tenth minute of exercise was 1.56 per cent.

Discussion

The results indicated a significant difference at the 5 per cent level of confidence, in all areas tested between smokers and non-smokers, therefore, the Null Hypothesis was rejected.

The finding agree with Reindell and Winterer[1] and Schlipp[2] that smokers had a higher resting pulse rate. The exercise pulse rate difference was very high and was probably due to many of the non-smokers being athletes while a majority of the smokers were not in a formal training program. However, it was of interest to note that at the end of the first exercise minute the pulse rate for smokers and non-smokers was almost the same. This meant the non-smokers had more of an increase in pulse rate during the first exercise minute. From this point the non-smokers pulse rate did not increase as rapidly as

[1] Reindell and Winterer, pp. 228-287.

[2] Schlipp, pp. 439-445.

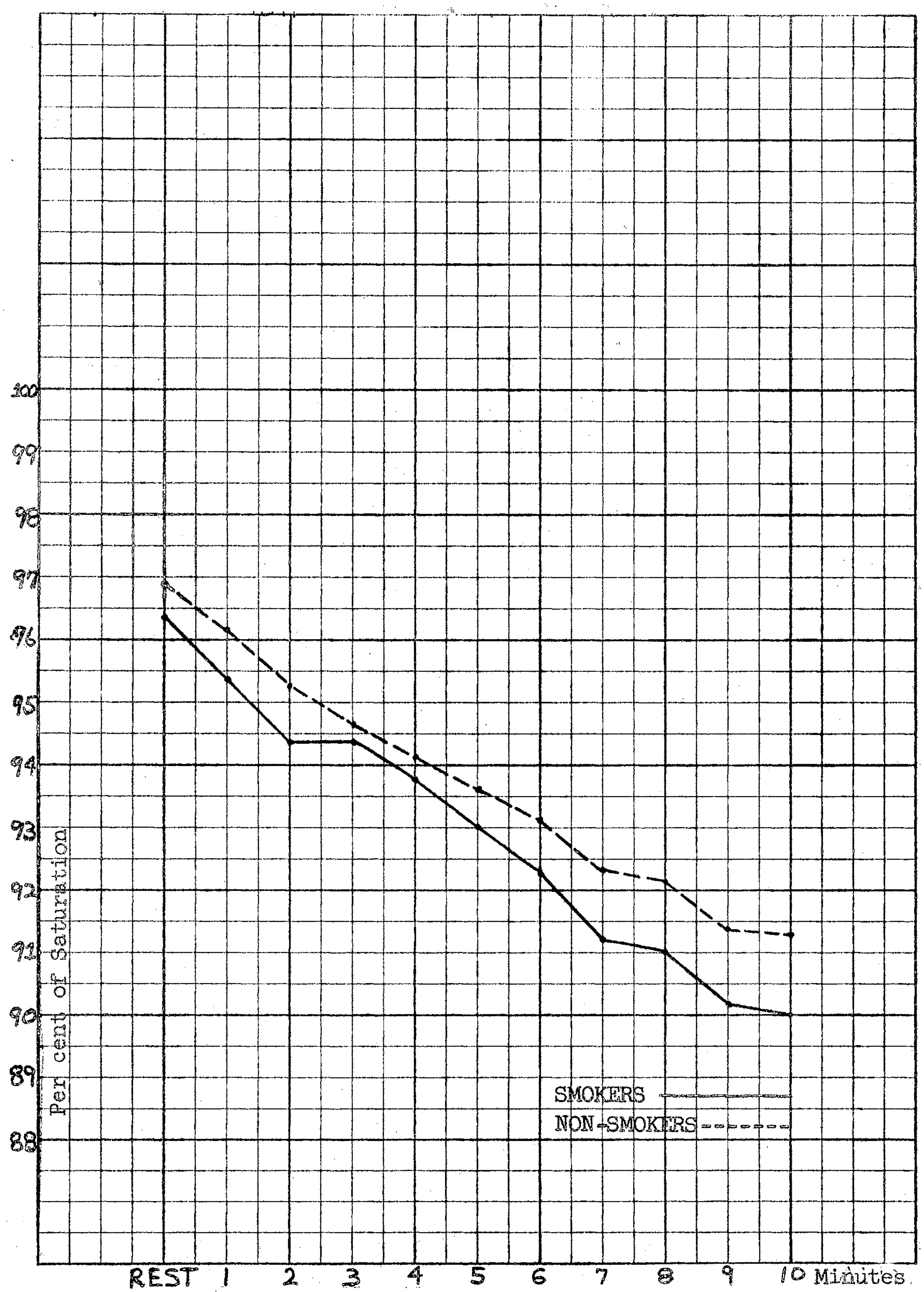

Figure 4. Oxygen Saturation Level at Rest and During Exercise

the smokers. This lower pulse rate level during the remaining exercise period indicates the non-smokers did not have to work as hard as the smokers which agrees with findings of Fodor, Glass and Weiner.[3]

The resting oxygen saturation level disclosed a lower reading in smokers than in non-smokers. This result agreed with the findings of McFarland[4] concerning carbon monoxide, but not to the same degree. It appeared that the presence of carbon monoxide in the blood was probably the main factor in the lower resting oxygen saturation level in the smoking group as found in the study by Chevalier.[5] The greater difference in the exercise oxygen saturation level may have been due to smokers having less access to oxygen caused by a decrease in airway conductance as shown by Nodel and Comroe.[6] Also less oxygen was available because of the carbon monoxide factor. Thus, less air and less oxygen carrying capacity together with at least as much oxygen consumption would create a greater deficiency in exercise oxygen saturation levels of smokers at the end of the exercise period.

The decrease in oxygen supply to the smokers brought about an increase in work load and this in turn causes the increase in pulse rate. The statement by Gibbons,[7] the work of McFarland,[8] and the results of this study show that the smokers have a distinct disadvantage during exercise when compared with non-smokers.

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[3]Fodor, Glass and Weiner, pp. 122-127.

[4]McFarland, pp. 417-443.

[5]Chevalier, pp. 1061-1064.

[6]Nodel and Comroe, pp. 713-716.

[7]The Daily Oklahoman, p. 4.

[8]McFarland, p. 417-433.

CHAPTER V

SUMMARY AND CONCLUSIONS

Summary

The purposes of the study were to determine if a difference existed between smokers and non-smokers in the resting oxygen saturation level, if exercise had an effect on oxygen saturation level, and if a relationship existed during and after exercise and a work load as indicated by pulse rate.

The literature contained many references on the effects of smoking and its relationship to oxygen use by the body, but a published reference was not found directly dealing with the purposes of the study.

All areas of comparison between smokers and non-smokers resulted in differences which were statistically significant at the 1 per cent level of confidence in favor of the non-smokers. The change in oxygen saturation level of the whole group from rest to post exercise showed a decrease which was statistically significant at the 1 per cent level of confidence.

The correlation between the lowest oxygen saturation level and the highest pulse rate for each subject was significant at the 1 per cent level of confidence. The group mean correlation between oxygen saturation levels and pulse rates at the end of the ten minute exercise period was significant at the 5 per cent level of confidence.

Conclusions

The results of this study warrant the following conclusions in regards to the subjects tested.

1. The resting pulse rates were significantly lower for the non-smoking group than for the smoking group.

2. The resting oxygen saturation levels were significantly higher for the non-smoking group than for the smoking group.

3. The exercise pulse rates were significantly lower for the non-smoking group than for the smoking group.

4. The exercise oxygen saturation levels were significantly higher for the non-smoking group than for the smoking group.

5. The increase in pulse rate indicated a significant negative correlation existed between oxygen saturation levels during and after exercise and workload as indicated by the increase in pulse rate and the decrease in the oxygen saturation levels measured for the combined groups.

Recommendations

1. It is recommended that additional work of this nature be done with a randomly selected sample with smokers stratified according to amount and length of time smoked to give a more complete picture of the effects of smoking on oxygen saturation levels. Measurement of blood carbon monoxide levels and airway conductance would add valuable information to such a study.

2. A study of the recovery rate of oxygen saturation levels in relation to the recovery rate of pulse rate would add to the knowledge of the effects of smoking on the body.

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APPENDIX A

RAW DATA - SMOKERS PULSE RATE

Subject	Rest	Minutes of Exercise									
1	78	138	156	156	162	162	162	162	162	162	168
2	68	126	132	132	132	132	132	138	138	144	150
3	78	124	138	144	156	162	162	162	168	168	174
4	66	102	108	114	114	120	126	132	132	132	132
5	72	114	138	138	138	156	156	156	162	162	162
6	78	138	144	150	156	156	162	162	162	162	162
7	84	150	150	156	156	156	156	162	162	162	168
8	84	138	150	150	156	156	162	162	162	168	168
9	72	96	132	144	150	150	150	156	156	156	156
10	72	132	144	150	156	162	168	168	174	174	174
11	72	120	138	138	138	138	138	138	144	144	144
12	78	126	132	132	138	144	144	144	150	156	156
13	78	114	132	150	150	162	162	162	162	162	162
14	66	108	108	114	114	114	126	132	132	132	132
15	78	126	132	132	132	138	138	144	144	144	144

APPENDIX B

RAW DATA - NON-SMOKERS PULSE RATE

Subject	Rest	Minutes of Exercise									
1	66	120	126	126	126	126	126	132	132	132	132
2	72	135	149	150	162	162	162	162	168	168	168
3	78	150	162	162	162	168	168	168	168	168	174
4	60	126	132	132	136	136	138	138	138	144	144
5	54	120	120	126	132	132	132	132	132	136	136
6	66	114	114	126	126	132	138	138	144	144	144
7	72	120	126	132	132	132	138	144	144	150	150
8	66	126	132	144	150	150	150	150	150	150	150
9	60	126	126	126	120	120	126	126	126	126	126
10	66	132	138	138	138	144	144	144	144	150	144
11	72	108	114	114	114	108	108	114	114	120	120
12	74	138	144	150	156	156	156	156	156	162	162
13	72	114	120	120	126	126	126	126	132	132	138
14	66	108	108	114	114	120	120	120	120	120	120
15	54	90	102	108	108	108	108	108	114	114	114
16	72	138	144	156	156	156	156	156	162	162	162
17	66	108	114	114	120	126	126	138	138	144	144

APPENDIX C

RAW DATA - SMOKERS - OXYGEN SATURATION LEVEL

Subject	Rest	Minutes of Exercise									
1	96.6	96.2	94.5	94.5	93.5	92.9	92.5	92.2	91.9	91.6	91.2
2	96.4	95.4	94.0	94.0	93.8	92.4	91.2	90.5	89.9	89.9	89.5
3	95.8	95.3	94.7	94.7	94.4	93.7	92.9	91.8	90.8	90.1	90.0
4	96.4	95.2	93.9	93.9	92.9	92.0	91.6	90.8	90.4	90.4	90.0
5	96.8	96.5	94.9	94.9	94.2	93.8	93.0	92.2	91.5	90.8	90.2
6	96.7	96.1	95.9	95.9	95.1	94.5	93.9	93.4	92.7	92.0	91.1
7	96.4	96.0	94.5	94.5	94.0	92.5	91.8	91.5	90.4	90.2	90.1
8	96.3	95.6	94.1	94.1	93.8	92.9	91.8	91.0	90.5	90.0	89.7
9	96.5	95.5	94.0	94.0	94.0	93.5	93.0	91.0	90.5	90.0	89.5
10	96.0	95.5	93.0	93.0	92.0	91.8	91.3	90.2	90.2	89.4	89.0
11	97.6	96.0	94.8	94.8	94.2	94.0	93.5	93.0	93.5	92.0	91.2
12	96.6	96.4	95.0	95.0	94.7	94.0	93.8	93.1	92.0	91.6	91.0
13	96.7	96.2	94.8	94.8	93.2	92.4	91.7	90.7	90.1	89.5	89.3
14	96.1	95.4	93.7	93.7	93.0	92.6	92.0	91.6	91.0	90.5	90.1
15	95.8	95.0	94.0	94.0	93.9	92.5	91.8	91.0	90.5	90.0	89.2

APPENDIX D

RAW DATA - NON-SMOKERS - OXYGEN SATURATION LEVELS

Subject	Rest	Minutes of Exercise									
1	97.4	96.4	95.3	94.7	94.3	94.0	93.9	93.2	93.0	92.8	92.2
2	97.3	96.5	95.0	94.0	94.0	93.5	92.0	91.5	91.0	90.5	90.0
3	97.2	96.0	94.3	93.5	92.5	91.7	91.2	91.0	90.8	90.4	90.2
4	97.6	97.3	97.0	96.7	96.4	96.0	95.8	95.4	94.1	93.8	93.2
5	97.3	96.4	96.0	95.9	95.5	95.1	95.0	95.0	94.6	93.8	93.4
6	97.2	95.7	95.0	94.0	93.8	93.2	93.0	92.7	92.2	92.0	92.0
7	97.3	96.4	95.8	95.0	94.2	93.6	93.0	92.5	92.0	91.8	91.6
8	97.6	96.3	95.3	95.0	94.4	94.0	94.0	93.8	93.2	92.9	92.6
9	97.2	95.8	95.6	94.6	93.8	93.8	92.9	91.8	91.2	91.0	90.8
10	97.9	96.5	96.3	95.8	94.9	94.6	93.5	92.5	91.3	91.0	90.7
11	96.9	96.0	95.1	94.0	93.2	93.0	93.0	92.9	93.0	93.2	93.2
12	97.8	96.8	95.4	94.7	94.0	93.4	92.6	91.8	91.0	90.6	90.2
13	97.3	95.9	94.8	94.5	93.2	92.4	92.2	92.0	91.8	91.8	91.9
14	97.2	96.0	94.8	94.2	94.0	93.4	93.0	92.6	92.2	91.8	91.6
15	97.2	96.0	95.4	95.0	94.0	93.8	93.0	92.5	92.3	92.0	91.0
16	97.3	95.2	94.5	93.5	92.9	92.5	92.2	91.9	91.6	91.5	91.5
17	97.0	96.3	96.0	95.3	95.0	94.6	94.1	93.6	92.8	92.0	91.6

APPENDIX E

FORMULAS

For t - ratio calculations: Standard t [1]

$$\sigma = \sqrt{\frac{\Sigma X^2}{N} - M^2}$$

$$\sigma = \frac{\sigma}{\sqrt{N-1}}$$

$$\sigma \, diff = \sqrt{\sigma M_1^2 + \sigma M_2^2}$$

$$t = \frac{M_1 - M_2}{\sigma \, diff}$$

For t - ratio calculation Dwyer t - [2]

$$t^2 = \frac{\Sigma X^2 (N-1)}{N \Sigma X^2 - (\Sigma X)^2}$$

$$t = \sqrt{t^2}$$

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[1]A.T. Slater-Hammell, "Computational Design for Evaluating the Significance of a Difference Between Means," Research Quarterly, XXXVI (1957) p. 212.

[2]Benton J. Underwood, et. al., Elementary Statistics (New York, 1954), p. 61.

For calculation of correlations:

$$r = \frac{\frac{\Sigma XY}{N} - M_x M_y}{\sigma_x \sigma_y}$$

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