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FROM MINUTE VENTILATION AT VARYING WORKLOADS

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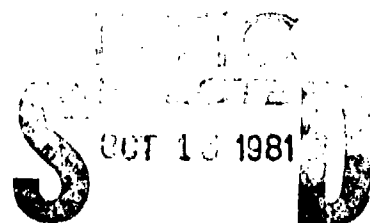
**A NOMOGRAM FOR CALCULATION OF OXYGEN CONSUMPTION FROM
MINUTE VENTILATION AT VARYING WORKLOADS***

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ABSTRACT

Oxygen consumption can be difficult and time consuming to measure. At the present time there does not exist an accurate method of estimating oxygen consumption during exercise at varying workloads. This study examined the relationship between minute ventilation and oxygen consumption and from the results generated a nomogram which can be used to accurately estimate oxygen consumption from minute ventilation in normal athletic male subjects. It was also determined that although a positive smoking history did not effect the accuracy of this estimate such a history adversely effects the ability to predict maximal oxygen consumption from heart rate at sub-maximal work rates.

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A Nomogram for Calculation of Oxygen Consumption from Minute Ventilation at Varying Workloads

T. S. Neuman and K. M. Moser

Exercise testing is frequently employed to evaluate different aspects of cardiopulmonary function (1). One of the principal measurements used to quantify response to exercise is the oxygen consumption. The direct measurement of oxygen consumption is time consuming and cumbersome. Unfortunately, no reliable indirect method for estimating oxygen consumption during exercise is now available. Minute ventilation has been shown to correlate highly with oxygen consumption at a given workload and during recovery from that workload (2). Previous reports also have noted a relationship between heart rate (HR) and oxygen consumption (3, 4, 5), and between minute ventilation and oxygen consumption (6, 7). Unfortunately statistical analyses have not been performed to assess whether oxygen consumption can accurately be estimated from HR or minute ventilation throughout graded workloads. This study was undertaken to determine whether HR and/or minute ventilation can be used to accurately predict oxygen consumption in well-trained, normal athletic subjects.

METHODS

Subjects. Sixty well-trained athletic male subjects, (age 19-34 years, mean = 28 ± 5.0 s.d; weight 58.5 - 102.5 kg, mean = 78.4 ± 10.4 kg; height 162-200 cm, mean = 179 ± 7.4 cm), who volunteered for this experiment were studied. 34 were non-smokers (less than one-half pack per year per lifetime); 16 were smokers; and smoking history was not available in 10. Informed consent was obtained from all of the subjects.

Design. To eliminate the possibility of occult lung disease, routine spirometry was initially performed. This consisted of the measurement of standard flows and volumes: maximal mid expiratory flow (MMEF), forced expiratory volume on 1 second (FEV1), vital capacity (VC), inspiratory reserve volume (IRV), and forced vital capacity (FVC). Functional residual capacity (FRC), residual volume (RV), total lung capacity (TLC), and airway resistance (RAW) were measured in a Collins variable pressure body plethysmograph. All subjects were additionally screened for the presence of any cardiorespiratory symptoms.

Following this evaluation the subjects completed a regimen of graded exercise (Astrand) (8) on a Collins bicycle ergometer with continuous electrocardiographic monitoring. The subjects (pedalling at 50 cpm) performed at the following workloads in sequence or until they were exhausted: 50, 100, 125, 150, 175, 200, 250, 300, 350, and 400 watts. The subjects exercised for a total of 5 minutes at workloads of 50 through 200 watts and for 3 minutes at 250 to 400 watts. A 15 minute rest interval was allowed in between each work period. Expired gases were collected during the last minute of exercise although gas collections were shortened during the final workload to 30 seconds if necessary (due to large $\dot{V}_E$). The expired gases were collected using a Collins triple valve and low resistance tubing. The volume of the expired gas was measured in a Tissot bell. $F_{E\text{CO}_2}$ and $F_{E\text{O}_2}$ were measured on a Goddard NV capnograph and Beckman E_2 oxygen analyzer, respectively, from aliquots of the mixed expired gas. Verification was accomplished with standard calibration gases and an IL 113 pH and gas analyzer. Oxygen consumption was calculated at STPD and correlated with

HR (obtained from the exercise electrocardiogram) and minute ventilation. $\dot{V}O_2$ was plotted against HR and $\dot{V}_E$. Oxygen consumption and $\dot{V}_E$ were expressed as liters/min, ml/kg/min, ml/meter/min, and ml/m²/min. HR was expressed as beats per minute and percentage of age adjusted maximum, (calculated both from $200 - \text{age}$ and $210 - .85 \text{ times age}$). These results were then plotted normally and semilogarithmically. Correlations between $\dot{V}O_2$ and the two measures of HR were computed and the mean square errors of prediction for the smokers and non-smokers contrasted by a one way analysis of variance. A t-test for comparison of correlated correlations was used to contrast HR - $\dot{V}O_2$ and $\dot{V}_E$ - $\dot{V}O_2$ correlations and a z-test between pairs of correlations was used to contrast the correlations between the smokers and non-smokers. (Both of these methods require a Fischer Z transformation of the correlation coefficient.) (17).

RESULTS

The preliminary pulmonary function testing revealed that the subjects were all normal as judged by the measured parameters. There were no statistically significant differences between smokers and non-smokers and multiple regression analysis did not indicate any combination of factors that differentiated smokers from non-smokers. Correlation coefficients between $\dot{V}O_2$ vs $\dot{V}_E$ and $\dot{V}O_2$ vs HR were highest when $\dot{V}O_2$ and $\dot{V}_E$ were expressed in ml/kg/min and when HR was expressed in beats per minute.

The relationship of $\dot{V}_E$ (ml/kg/min) to $\dot{V}O_2$ (ml/kg/min), plotted normally, resulted in a correlation coefficient of .936 ($p < 10^{-5}$); when plotted semilogarithmically, the r was 0.959 ($p < 10^{-5}$).

For the relationship of HR (beats/min) to $\dot{V}O_2$ (ml/kg/min), plotted

normally, the correlation coefficient was 0.817 ($p < 10^{-5}$); when plotted semilogarithmically, r was 0.806 ($p < 10^{-5}$).

When smokers and non-smokers are analyzed separately for the individuals on whom data are available ($n=50$), the correlation coefficients for the relationship between $\dot{V}_E$ and $\dot{V}O_2$ were not significantly different ($r = .947$ and $.954$ for smokers and non-smokers respectively). Separating smokers and non-smokers the plot of HR vs $\dot{V}O_2$ results in a correlation coefficient of $.828$ for the smokers and $.853$ for non-smokers: thus the correlation coefficient for $\dot{V}_E$ vs $\dot{V}O_2$ is significantly higher than that for HR vs $\dot{V}O_2$ for both smokers ($t = 7.54$, $p < 10^{-5}$) and non-smokers ($t = 10.15$, $p < 10^{-6}$). Although not statistically significant, the slope of the plot of HR and $\dot{V}O_2$ in the smokers is higher ($m = 2.15$) than in the non-smokers ($m = 2.02$), indicating that the mean HR of smokers is higher than non-smokers during submaximal exercise for any $\dot{V}O_2$.

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Fig. 2 is a nomogram prepared from the data in Fig. 1 which can be used to estimate $\dot{V}O_2$ based upon measurements of weight and minute ventilation at any workload. If comparisons are made between actual $\dot{V}O_2$ and predicted $\dot{V}O_2$ the standard error of the estimate (prediction) is 4.08 ml/kg/min using $\dot{V}_E$ and 7.47 using HR. Thus there is almost two-fold reduction in the error obtained if $\dot{V}_E$ is used to estimate $\dot{V}O_2$.

DISCUSSION

These results confirm previous reports that $\dot{V}O_2$ and minute ventilation are highly correlated (6, 7). It has been recognized that the linear nature of this relationship at low workloads deteriorates at higher workloads (9). By normalizing the measured parameters (of $\dot{V}O_2$ and $\dot{V}_E$

to body weight) and by plotting them in a semilog fashion, the relationship now accurately predicts $\dot{V}O_2$ at any level of exercise in the test group. It must be emphasized that the test group consists of well-trained athletic men aged 19-34; before these data are extrapolated to other groups, verification of their validity in those groups would be necessary. Of note, however, is smokers and non-smokers were included within the test group. Smoking history did not influence the relationship between $\dot{V}_E$ and $\dot{V}O_2$, but the correlation of HR and $\dot{V}O_2$ increased from .817 in the whole group to .853 in the non-smokers. This implies that smoking might somehow effect the HR response to exercise; since the slope of HR vs $\dot{V}O_2$ is higher in smokers than non-smokers, it indicates that, during submaximal exercise, the mean HR of smokers is higher than non-smokers. This has been suggested by previous work (10, 11). These differences cannot easily be explained by broncho-pulmonary disease, as pulmonary function testing and clinical screening did not reveal the presence of any pathology.

Although maximal oxygen consumption does not change in normals who smoke (10, 11, 12, 13), HR at submaximal exercise may. Thus previous studies that use HR during submaximal exercise to extrapolate to maximal oxygen consumption should be re-examined so that the effect of smoking history is considered.

We have shown that minute ventilation is capable of predicting oxygen consumption extremely accurately at a given workload. As a result, measured HR, and oxygen consumption estimated from minute ventilation at that workload, can be used to predict maximal oxygen consumption from already published nomograms (14, 15). Additionally, oxygen consumption can be easily estimated at more than one workload allowing a

more accurate extrapolation to maximal oxygen consumption than can be obtained from a single workload measurement (16), although as suggested by our results the possible effects of smoking still need to be evaluated.

In conclusion, we have made the observation that $\dot{V}_E$ correlates highly with $\dot{V}O_2$ in normal athletic males irrespective of smoking history and we have constructed a nomogram that can be used to predict $\dot{V}O_2$ from $\dot{V}_E$ during exercise for this group. Single or multiple determinations of $\dot{V}E_2$ can then be used to predict maximum oxygen consumption without measuring $\dot{V}O_2$. Furthermore, we conclude that the correlation of $\dot{V}O_2$ and HR may be influenced by smoking and as a result suggest that studies noting the influence of smoking on prediction of maximal aerobic capacity be performed.

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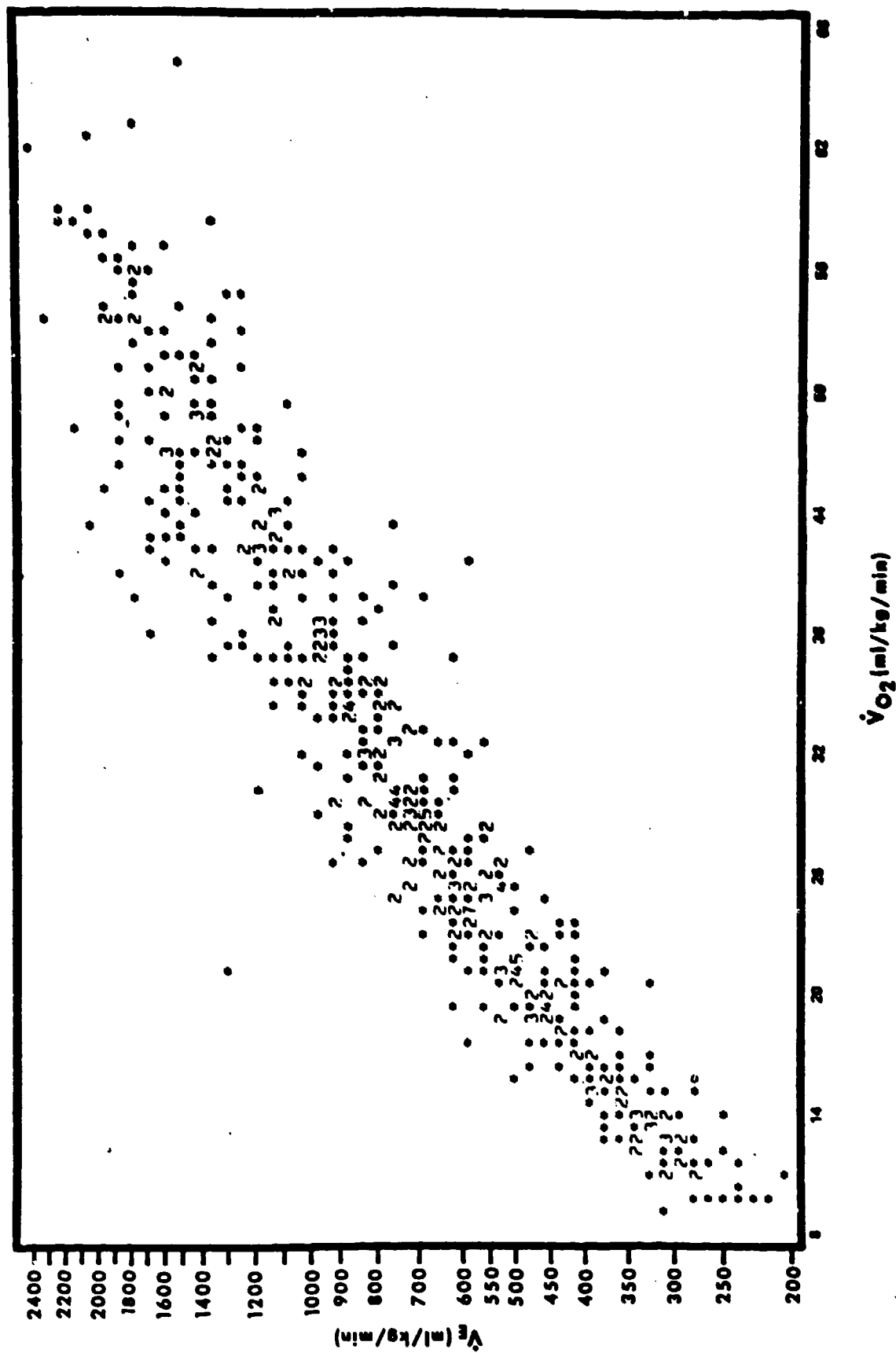


Fig. 1. Graph of $\dot{V}_{O_2}$ (ml/kg/min) and $\dot{V}_g$ (ml/kg/min). Asterisks represent single datum points and numbers (2,3,4,5) represent multiple data points.

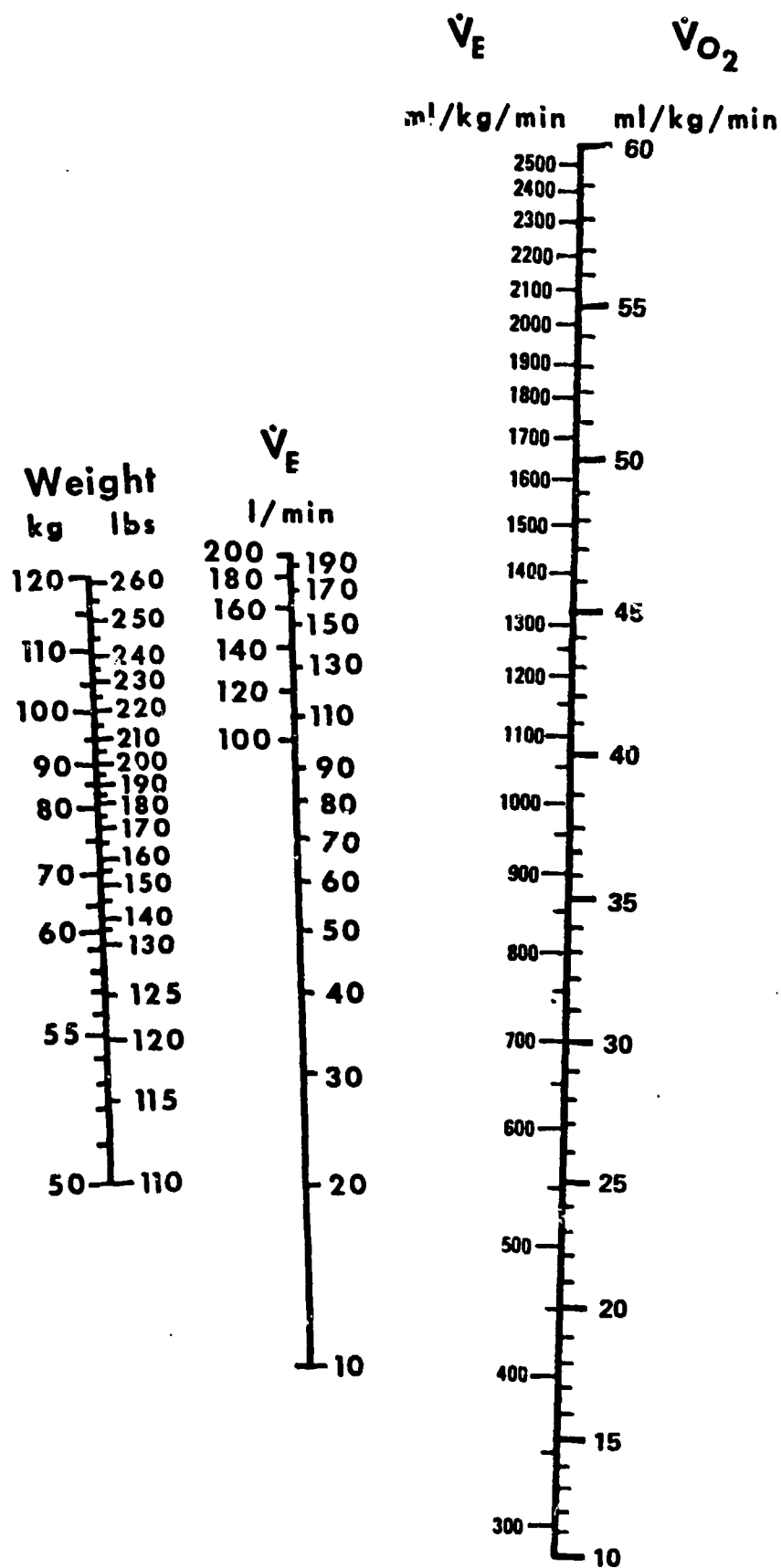


Figure 2. Nomogram for the calculation of $\dot{V}_{O_2}$ (ml/kg/min) from weight (kg or lbs) and $\dot{V}_E$ (l/min).

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