

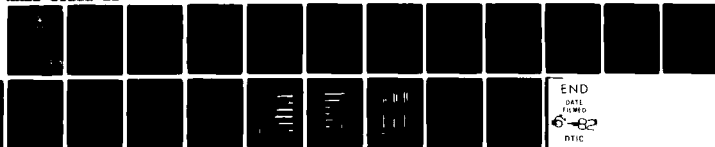
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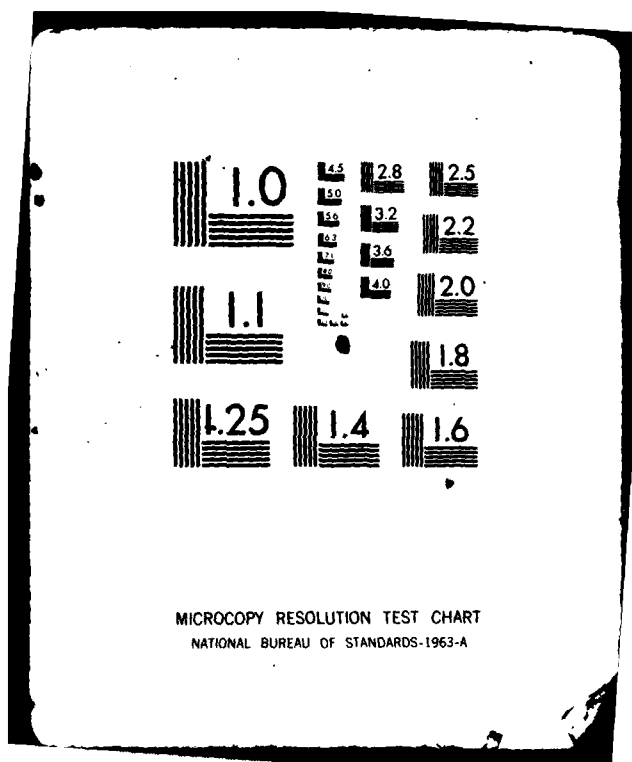
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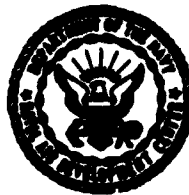
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**PULMONARY FUNCTION MEASURES BEFORE AND AFTER EXPOSURE OF
HUMAN SUBJECTS TO $+G_z$ AND $+G_x$ ACCELERATION LOADS**

Edwin Hendler
Aircraft and Crew Systems Technology Directorate
NAVAL AIR DEVELOPMENT CENTER
Warminster, Pennsylvania 18974

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INTRODUCTION

The fact that directing a sustained G vector through the transverse, rather than along the longitudinal axis of the body, will result in greatly increased G tolerance has been known for about 50 years (reference 1). In the decade following World War II, there was considerable laboratory activity by investigators attempting to exploit this knowledge (references 2, 3, and 4), including installation of a supine seat in a Naval aircraft which was then evaluated in flight (reference 5). To counter objections regarding the reduction in forward vision following supination, von Diringshofen in 1955 described what he called the "long chair position" (reference 6). He showed how the location of the pilot's head and neck could be maintained unchanged while his torso and legs were raised. Using this procedure, the pilot's eye position could be kept at the same location in the cockpit as when he was seated upright, the vertical distance between the heart and brain could be reduced, and venous return to the heart from the lower body could be enhanced. von Diringshofen speculated that by using his proposed "long chair position" the pilot would be able to tolerate exposures to 3G for long durations, without experiencing visual disturbances. A resurgence of interest in the usefulness of the supinated body position to increase G tolerance has occurred during the past decade. This interest has been stimulated by the development of very high performance aircraft, such as the F15, F16, and F18, which can perform maneuvers generating G levels appreciably above those tolerated by the upright seated pilot, even when he is protected by an inflated anti-G suit and is performing the M-1 maneuver.

In both the upright and supine body positions, the lungs have been shown to be particularly susceptible to changes in applied G loads. Unlike any other tissues in the body, the pulmonary alveoli remain at one atmosphere pressure during G exposure, while the pressure of the blood bathing them increases directly in proportion to the applied G load. This unbalance of pressures is potentially disruptive to the delicate air-containing network making up the structure of the lung. In addition the balance between pulmonary ventilation and pulmonary blood flow (perfusion) is easily upset, resulting in alterations in gas exchange capable of affecting many tissues and organs of the body. Basic to proper respiration are the mechanical movements by which the lungs are ventilated; both the direction of the G vector and its magnitude alter the ventilatory capability of the individual.

The study to be described was conducted in conjunction with a long term investigation of the psychophysiological effects of repeated exposure to accelerations of tolerable levels. Since both the upright and supine body positions were to be used by subjects exposed to acceleration patterns produced by the NAVAIRDEVCON Dynamic Flight Simulator (DFS), or human centrifuge, it was decided to utilize available equipment to measure some aspects of dynamic lung volumes at various times during the experimental procedures. As expected, it was found that both changes in body position and the presence of an inflated anti-G suit (AGS) reduced measured lung volumes and respired gas flows. For the conditions studied, no significant changes in dynamic lung volumes were measured which persisted after exposure to the acceleration patterns.

METHOD

Routine measures of dynamic lung volumes were obtained on a group of four male subjects who had volunteered to participate in the last two phases of an extended study of repeated acceleration exposure effects. The informed consent of the subjects was obtained and the proposed procedures were reviewed by the NAVAIRDEVCON Committee for the Protection of Human Subjects, in accordance with SECNAV Instruction 3900.39A of 20 March 1978. The physical characteristics of these subjects are given in table I. Table II shows baseline evaluations on selected pulmonary function measures. All baseline measures were made using a fully automated pulmonary function

screening system* consisting of a wedge spirometer, an electronic interface unit, and a Tektronix Model 4051 BASIC graphic computing system**. In phase I of the study, each of the subjects was thoroughly coached in the procedures to be followed and performed respiratory maneuvers to obtain records of slow vital capacity (SVC), maximum voluntary ventilation (MVV), and forced vital capacity (FVC). From these three, all of the pulmonary function measures listed in table II† were derived automatically and a comparison of values obtained was then made with predicted values. The latter resulted from the solution of empirically derived equations which had been selected from the scientific and clinical literature (references 8 and 9) and stored in computer memory. Based on the subject's sex and age group, appropriate equations from among those stored were selected and solved automatically for each of the pulmonary function measures using the subject's age and height. In printing out the results, measured and predicted values were listed, as well as the percentages of the predicted represented by the measured values.

While performing the baseline tests, the subjects were dressed in shirt, trousers, and socks, shoes, and underwear. In phase II of the study, they donned summer flight coveralls to replace their shirts and trousers, as well as individually fitted MA-2 torso harnesses and MA-2A suits; bladders of the AGS were inflated to 2 psig for some of the baseline FVC tests to be described. Baseline FVC testing was conducted under four conditions: 1) Seat back angle (SBA) = 15°, AGS uninflated; 2) SBA = 15°, AGS inflated; 3) SBA = 75°, AGS uninflated; and 4) SBA = 75°, AGS inflated. A demonstration PALE (Pelvis and Legs Elevating) articulating seat was utilized, the seat back angle of which could be adjusted to the values indicated (reference 7). Each subject performed a series of three forced vital capacities, with a suitable resting period separating successive efforts. The pulmonary function screening system was arranged to present volume and flow data derived from tidal volume and forced vital capacity maneuvers in the form of flow-volume (\dot{V} - V) loops, an example of which is shown in figure 1. Included in this figure are numerical values showing actual, predicted, and percent values of pulmonary function measures derived from the automated analysis of the FVC provided by the testing system. Table III shows the measures derived from the V - V loops under the four conditions described above.

Because of stringent limitations on available space, the pulmonary function screening system could not be used when the subjects were seated in the DFS gondola. As a compromise, a portable respirometer consisting of a pneumotachograph and pressure transducer mounted in a 20 in. long metal tube as shown in figure 2 was used. The use of the portable respirometer constituted phase III of the study.

The purpose of the metal tube mentioned above was to enhance laminar flow and to act as a heat exchanger, cooling the expired gases before they passed through the pneumotachograph. A bracket mounted in the gondola supported the portable respirometer in a location accessible to the seated subjects. Because of his size, subject 2 was unable to perform the required respiratory maneuvers in using the portable respirometer when he was placed in the supinated (SBA = 75°) position. The portable respirometer was employed before and after each subject's exposure to the acceleration patterns described below; the electrical output from the respirometer was fed directly into the interface and Tektronix units of the pulmonary function screening system which was positioned on a platform adjacent to the gondola. In essence, the portable respirometer was used as a replacement for the wedge spirometer††.

*Manufactured by S&M Instrument Company, 59 Pine Valley Rd., Doylestown, PA 18901.

**Manufactured by Tektronics, Inc., 14150 S.W. Karl Braun Dr., Beaverton, OR 97077.

†The caption of Table II includes a listing of all the pulmonary function measure abbreviations and their meanings.

††Because of technical difficulties, no respiratory mechanics measures were obtained on the second day of the DFS runs.

The schedule for acceleration exposure extended over an 8 day test period. Before the test period began, the subjects were trained for several weeks to perform tracking and response time tasks in the upright and supinated body positions; they received both static and dynamic training sessions in the DFS. All of the subjects had previous experience riding the DFS, including high G exposures during which they wore restraints and the AGS and performed the M-1 maneuver.

Each subject received one acceleration *exposure* daily, consisting of six consecutive *runs*. One minute rest periods separated each of the runs. Two haversine-shaped G pulses (acceleration pulses), with constant onset and offset durations of 4s each, were applied in each run; the pulses had plateau durations of either 20 or 40s and were separated from each other by 4, 8, or 32s. Both of the acceleration pulses in any given run were identical in all respects. Run conditions are presented in table IV. The combinations of conditions for all runs were selected on the basis of their being below the tolerance limit of the subjects. In addition to the garments worn during the baseline FVC testing, electrodes were attached to the skin of the subjects for the collection of biomedical data. AGS inlet pressures were regulated by a specially designed and constructed electronic valve which was adjusted to start pressurization at 1.5G and linearly increase pressure with G until the final pressures shown in table IV were attained. At the termination of the G plateau, suit pressure decreased linearly with G, so that no residual pressure remained by the time the 1.5G level was reached.

RESULTS

An examination of table II shows the functional status of the lungs and airways of the individuals serving as subjects in this study. Measured lung volumes and capacities can be seen to be well within the normal (predicted) range for all subjects, suggesting the absence of restrictive pulmonary impairment. This impression was supported by the physical examinations of the subjects and a review of their medical histories. On the other hand, the data measuring dynamic changes dependent upon the rapid movements of gases in and out of the lungs suggest an obstructive impairment in both subjects 1 and 2. For these two subjects, MMEFR is clearly below normal levels and FEV_1/FVC ratios are under the normal lower limit of 80 percent. In addition, MVV for subject 2 is noticeably reduced and when the difference between FVC and SVC is calculated, the value of 430ml for this subject far exceeds the normal upper limit of 250ml for trapped air. In conformance with the reduced inspiratory and expiratory flows shown for subject 2 in table II, his $\dot{V} - V$ loops consistently exhibited the squared-off configuration typical of those obtained from individuals with obstructive pulmonary disease.

For those measures (FVC, FEV_1 , FEV_3 , FIVC, MMIFR, and MMEFR) which were common to the baseline tests conducted in the upright seated position before (phase I) and after (phase II) the subjects had donned the torso restraint harness and uninflated AGS, all (except for MMIFR for subject 1) decreased after the subjects donned the gear mentioned. The decreases in individual measures ranged from about one-half to about 24 percent, with an overall mean decrease of about 6 percent. The largest decrease over all the subjects averaged 13 percent for FEV_1 . However, when the mean values for each of the pulmonary function measures were compared across all subjects, there was no significant difference between those made before the harness and AGS were donned and those made afterward.

In figure 3, the mean maximum values derived from baseline FVC testing (phase II) of the subject group are plotted with the conditions ordered from upright and without the AGS inflated to supine and with the AGS inflated. In most cases, there is a progressive decrease in each of the pulmonary function measures as SBA increases from 15° to 75°, either alone or with the AGS inflated.

Each of the test conditions was therefore rated as follows: 1) SBA = 15°, AGS uninflated; 2) SBA = 75°, AGS uninflated; 3) SBA = 15°, AGS inflated; and 4) SBA = 75°, AGS inflated, and the least squares fit of the mean maximum values for the six pulmonary function measures to these conditions was determined. R^2 as a measure of goodness of fit (reference 10) was calculated on the basis of linear regressions and the following values were obtained: FVC, $R^2 = 0.97$; FEV₁, $R^2 = 0.93$; FEV₃, $R^2 = 0.88$; MMEFR, $R^2 = 0.88$; FIVC, $R^2 = 0.86$; MMIFR, $R^2 = 0.96$. The two measures of flow, MMEFR and MMIFR, seem particularly sensitive to changes in the test conditions, as shown in figure 4, where the measures are given as percent changes from those where SBA = 15° with the AGS uninflated. In fact, for FEV₁, FEV₃, and FIVC, AGS inflation seems to show a greater effect than the change in seat back angle.

The means of measures made with the portable respirometer (phase III) for each subject (table V) are compared in figure 5 with corresponding means derived from phases I and II. Since all of the measures made with the portable respirometer were carried out with the AGS uninflated, only the effect of supination can be shown for phase III. The predominant effect is a decrease in the pulmonary function measures made using the portable respirometer, with the greater decrease occurring in the SBA = 75° condition. Most changes in the magnitude of the pulmonary function measures ascribable to exposure to the acceleration profiles in the DFS were insignificant, but the direction of change was rather consistent, with the measures made after exposure somewhat larger than those made before.

DISCUSSION

The selection of subjects for the study described here was based primarily on their familiarity with the acceleration environment and training on the two tasks designed to measure performance. All of the subjects had performed in a variety of prior studies in which exposure to acceleration tolerance levels was involved, and none had experienced any special difficulties relating to respiration or any other specific physiological processes. For these reasons, the two smokers with signs of pulmonary obstruction were not excluded from participation in this study. Except for the initial deficiencies cited with respect to some of the pulmonary function measures, there is no evidence from the data collected, from comments of the subjects, nor from observations by the medical monitors to show that subjects 1 and 2 responded in any unusual manner to the test conditions.

Of the conditions imposed on our subjects, those expected to have the greatest effect on respiration include the wearing of special clothing, the position of the body, and G_z and G_x loads. Consequently, these factors will be briefly discussed and pertinent relations to our findings will be mentioned. When the chest is strapped to mechanically restrict respiratory movements so that TLC (total lung capacity) is reduced by 37 percent, VC (vital capacity) by 44 percent, and FRC (functional residual capacity) by 32 percent, the elastic recoil pressure of the lung and the maximum expired flow rate, both measured at 50 percent of TLC, increase (reference 11). Various explanations have been proposed to explain these increases, which had also been observed by other investigators previously, including the possibility that distortion of the lung induced by chest strapping makes it necessary to exert greater transpulmonary pressures to distend the lung to a given volume. However, it has been reported that after strapping has been removed from the chest, subjects continue to show increased elastic recoil pressure and expired flow rates so long as they continue to breathe at low lung volumes; once a deep breath has been taken, normal characteristics of the lung are reestablished (reference 12). Although the torso harness and AGS assemblies used in our study were individually fitted and adjusted, they were not tightened to the point where any discomfort resulted. The torso harness in particular is constructed in such a manner that, while it fits the torso snugly, it appears to distribute pressure much more evenly over the parts it covers than do chest straps which have been described in the literature. In contrast to the reductions in lung volumes of 30 to 40 percent

described by those using chest straps (references 11 and 12), our subjects shows mean decreases of 2.5 percent in FVC and 1.7 percent in FIVC by donning the torso harness and AGS. Therefore at the relatively expanded lung volumes over which our pulmonary function measures were made, there is no reason to expect that lung recoil pressures or flow rates would show increases, such as those mentioned above. In fact, while decreases in pulmonary function measures were consistently demonstrated after donning the torso harness and AGS, the changes involved were relatively small and not statistically significant.

Changes from the supine to the erect body positions, and the reverse, cause important shifts to occur in the relative positions of many of the body organs and in the distribution of gases and liquids contained within the body walls. Because of its size, composition, and close proximity to the chest, the abdomen as a fluid-filled, flexible walled container exerts a major influence on the process of respiration. Since the chest and abdominal cavities are separated by the flexible sheet of muscles making up the diaphragm, it can readily be appreciated that shifts in body position, or re-direction of the gravity vector, can result in intrusion of the contents of either of these cavities into the space ordinarily occupied by the other. In addition, pressure exerted on the walls of the chest or abdominal cavities, such as that resulting from expansion of the abdominal bladder of the AGS, also causes distortion of the usual cavity configuration with resultant displacement of contents and impingement into adjacent spaces. Since the compliance of the lung and chest wall are about equal, but opposite in sign (reference 13), intrusion of a substance into the chest cavity not only causes collapse of some lung tissue but also an expansion of the thorax. However, if the thorax is impeded from expanding by clothing or restraints, it would be expected that the volume of inflatable lung tissue would be reduced accordingly.

Reports of changes in lung volumes resulting from postural changes are numerous and characterized by their variability. Much of the latter can be ascribed to differences in techniques and subject samples examined. In general, however, when moving from the erect to the supine position, decreases in FRC, ERV, VC, and TLC have been measured, with small increases in IC (inspiratory capacity) reported. These volume changes have been ascribed to elevation of the diaphragm, increase in the transverse and anteroposterior diameters of the chest, and most importantly, to the influx of blood into the chest cavity. By measuring the volume of the trunk concurrently with lung volumes, Sjöstrand (reference 14) found that a mean volume increase of 440ml occurred in FRC with a change in body position from supine to erect, and he attributed this volume shift to blood leaving the chest cavity. Others have shown that if the capacity of the available extrathoracic vascular bed is reduced by placing tourniquets around the limbs, by immersion of the body in water, or by cooling peripheral areas to cause vasoconstriction, the blood volume leaving the thoracic cavity can be reduced when an individual changes from a supine to an erect posture.

Previous work (references 15 and 16) has shown that when a supinated individual is exposed to $+G_x$ loads of the magnitudes used in the present study, the resulting increase in weight of the anterior chest wall and abdominal viscera greatly impedes respiration and causes substernal discomfort and pain. VC, ERV, and FRC are noticeably reduced. On assuming an erect posture and being exposed to $+G_z$ loads, the consequent drop in VC is smaller and FRC increased somewhat, in comparison to the volume changes described for $+G_x$. Inflation of the AGS reduces VC further and reverses the increase in FRC. In addition, some of the small airways in the lung close off, starting at its base and moving upward with increasing levels of acceleration load. Ventilation-perfusion ratios range from infinity at the lung apex at $+3G_z$ (no perfusion) to 0.4 at the base (reference 17). There is a spatial ventilation gradient which normally exists in the lung, with ventilation increasing in alveoli from the apex to the base; the ventilation gradient is increased by acceleration and directed along the resultant acceleration vector (reference 17). Reflex changes induced in the systemic circulation during acceleration exposure are absent in the pulmonary circulatory system, and

pulmonary arterial pressure falls at heart level as applied $+G_z$ levels increase. Under $+G_z$ loads, blood flow to the upper parts of the lung is reduced and is increased to the lower parts. Similarly, under $+G_x$ loads, blood flow to the "upper" (anterior) parts of the lung is proportionately reduced while it increases to the "lower" (posterior) parts. For equivalent G levels, much more of the lung is deprived of blood when the vector is directed along the "X" axis than along the "Z" axis. Bronchodilatation, which is postulated to occur in the lower, hyperemic parts of the lung (reference 18), has been used to explain the mean increase of 23 percent in anatomic dead space measured in subjects exposed to $+5G_x$. Although alveolar pressure remains constant throughout the lung when the airways stay open during G exposure, pressure exerted on the lung is G dependent (estimated as 0.25 cm H₂O/cm/G), so that the lung is more compressed at its more dependent parts. Thus, Glazier et al (reference 19) showed that the average volume of apical alveoli in the lungs of dogs wearing an inflated abdominal bladder, exposed to $+3G_z$, and frozen *in situ*, was ten times greater than that of basilar alveoli. The G vector (both in magnitude and direction) is therefore the principle factor which affects ventilation in various parts of the lung. Even though the G vector may increase to appreciable levels, closure of small airways retains the air in many of the alveoli, thereby preventing them from collapsing to much below a fifth of their expanded volume. The closing volume represented by this air increases with G, reaching as high as 60 to 70 percent of VC at $+5G_x$ (reference 20). If pure oxygen is breathed instead of air (as would most probably be the case for pilots in an air combat situation), absorptional atelectasis could occur in the most dependent parts of the lungs, with resulting arterial desaturation. However, even when air is breathed, atelectasis can occur and is believed to have occurred fairly frequently in the present study, where coughing was often observed both during and immediately after G exposures.

There is little doubt that the conditions of acceleration exposure in our study produced changes in pulmonary function of the kinds described above. However, most of the changes were rapidly reversed, once the conditions producing them were removed. Gillingham and Krutz reported that after exposure of 11 subjects to 7 - 9 $+G_z$ for 45s, four of the subjects who were smokers showed a significant decrease in VC at 60 to 90s post-G (reference 21). Unfortunately, the time required to prepare and use the portable respirometer in our study was at least several minutes after the G level had returned to 1. Bush *et al* (reference 22) found that pulmonary resistance reached a maximum decrease 3 minutes after exposure of subjects to 5 and 7 $+G_z$ for 120s and 45s, respectively, and that 6 minutes and 12 minutes were required to return to pre-exposure values for the two G levels investigated. Both of these $+G_z$ exposures exceed the G-time combinations used in our study, so that changes in pulmonary resistance may have been more marked than those which probably occurred in our subjects. Our consistent finding of a small increase in the pulmonary function measures following the G exposures may indeed reflect a similar decrease in pulmonary resistance, a response which Bush *et al* attribute to a sympathetically mediated dilatation of the airways (reference 22).

The decreases in pulmonary function measures resulting from use of the portable respirometer are especially marked in the SBA = 75° position (figure 5). In addition to the expected decreases based on the factors previously discussed, such as the shift in body fluids, the arrangement of the portable respirometer within the DFS gondola may have involved additional restrictions which were not experienced by the subjects when they performed the pulmonary function tests in phase II. Unfortunately, it was not feasible to conduct a complete test series using the portable respirometer outside of the gondola. Such testing will be required in order to isolate the cause or causes of the changes observed.

The present study represents an initial attempt to measure dynamic lung volumes associated with the exposure of human subjects using G-protective measures in both the upright and supine body positions. It is planned to modify the portable respirometer to enable pulmonary function measures to be made either during exposure to acceleration loads or immediately following such exposure.

ACKNOWLEDGEMENT

The participation of HMC R. Rice, HM1 D. Murray, HM1 K. Hammergren, and PR2 J. Reed as volunteer subjects made this study possible.

Mr. J. Palumbo calibrated the portable respirometer, collected data using it, and modified the computer program of the automated pulmonary function screening system to make it suitable for use with the portable respirometer.

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TABLE II. Baseline Respiratory Measures in the Upright, Seated Body Position (Phase I).
(Meanings of abbreviations shown in footnote)

	SUBJECT 1	SUBJECT 2	SUBJECT 3	SUBJECT 4
FVC	4.22 (85)*	4.33 (89)	6.19 (99)	4.18 (86)
FEV ₁	3.10 (79)	2.93 (73)	4.85 (98)	3.60 (88)
FEV ₁ %VC	73	68	78	86
FEV ₃	3.86 (80)	3.90 (81)	6.02 (98)	4.12 (86)
FEV ₃ %VC	91	90	97	99
MMEFR	2.52 (47)	2.83 (51)	4.83 (77)	6.24 (110)
PEFR	8.40 (107)	9.44 (118)	11.00 (120)	10.72 (131)
FEF 75%VC	7.28 (88)	7.60 (91)	10.24 (112)	10.72 (128)
FEF 50%VC	4.00 (69)	4.24 (71)	7.20 (109)	8.60 (142)
FEF 25%VC	1.28 (45)	1.44 (46)	3.00 (81)	3.56 (106)
FIVC	4.12 (83)	4.05 (83)	6.06 (97)	4.32 (88)
MMIFR	4.02 (63)	2.45 (37)	4.62 (62)	8.21 (120)
FIF 75%VC	3.68 (78)	2.12 (45)	3.32 (62)	8.60 (179)
FIF 50%VC	4.36 (84)	3.36 (62)	4.12 (73)	9.12 (163)
FIF 25%VC	4.12 (56)	3.12 (45)	6.12 (90)	7.80 (118)
SVC	4.28 (87)	4.76 (97)	6.39 (102)	4.43 (90)
IC	2.71 (86)	3.59 (120)	4.24 (111)	3.69 (124)
ERV	1.57 (84)	1.17 (61)	2.15 (87)	0.74 (38)
MVV	141 (84)	109 (64)	223 (108)	164 (93)

FVC = Forced Vital Capacity (liters); FEV₁ = Forced Expiratory Vol. at 1 s (liters); FEV₁ %VC = Forced Expiratory Vol. at 1 s as a percentage of the Vital Capacity (%); FEV₃ = Forced Expiratory Vol. at 3 s (liters); FEV₃ %VC = FEV₃ as a percentage of the Vital Capacity (%); MMEFR = Mid-Maximum Expiratory Flow Rate (liters/s); PEFR = Peak Expiratory Flow Rate (liters/s); FEF = Forced Expiratory Flow at the percentage of the Vital Capacity shown (liters/s); FIVC = Forced Inspiratory Vital Capacity (liters); MMIFR = Mid-Maximum Inspiratory Flow Rate (liters/s); FIF = Forced Inspiratory Flow at the percentage of the Vital Capacity shown (liters/s); SVC = Slow Vital Capacity (liters); IC = Inspiratory Capacity (liters); ERV = Expiratory Reserve Vol. (liters); MVV = Maximum Voluntary Ventilation (liters/min)

*Values in parentheses are percentages of predicted values based on subject's age and height, as given in references (8) and (9).

TABLE III. Baseline FVC Pulmonary Function Measures Derived from Flow-Volume Loops (Phase II).
 Entries are mean and standard deviations, in parentheses. N = 3, except where noted.

	SBA = 15°				SBA = 75°				SBA = 15° + AGS				SBA = 75° + AGS			
	SUBJECT				SUBJECT				SUBJECT				SUBJECT			
	1**	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
FVC* (liters)	4.04 (0.10)	4.20 (0.21)	6.14 (0.04)	4.10 (0.13)	4.07 (0.06)	4.47 (0.04)	6.17 (0.02)	3.73 (0.19)	3.98 (0.12)	3.88 (0.22)	6.18 (0.03)	3.88 (0.33)	3.77 (0.02)	4.23 (0.12)	5.94 (0.03)	3.65 (0.29)
FEV ₁ (liters)	2.75 (0.44)	2.61 (0.49)	4.27 (1.00)	2.93 (0.61)	2.93 (0.03)	3.28 (0.26)	4.59 (0.11)	3.17 (0.02)	2.90 (0.08)	2.11 (0.13)	4.74 (0.09)	3.27 (0.20)	2.70 (0.03)	2.07 (0.30)	4.56 (0.12)	3.09 (0.51)
FEV ₃ (liters)	3.71 (0.04)	3.66 (0.40)	5.95 (0.11)	4.04 (0.10)	3.70 (0.03)	4.25 (0.06)	5.90 (0.06)	3.62 (0.19)	3.61 (0.06)	3.43 (0.22)	5.95 (0.03)	3.73 (0.30)	3.42 (0.04)	3.71 (0.08)	5.74 (0.06)	3.54 (0.31)
MMEFR (liters/s)	2.35 (0.38)	2.00 (1.09)	4.25 (0.97)	6.06 (0.32)	2.21 (0.09)	3.13 (0.40)	4.11 (0.13)	4.77 (0.18)	2.17 (0.05)	1.51 (0.18)	4.32 (0.10)	4.95 (0.45)	1.93 (0.12)	1.33 (0.29)	4.30 (0.40)	4.70 (0.76)
FIVC (liters)	4.10 (0.13)	3.89 (0.16)	6.00 (0.67)	4.26 (0.05)	4.06 (0.04)	3.97 (0.20)	6.04 (0.09)	4.20 (0.80)	3.92 (0.12)	3.29 (0.12)	5.79 (0.27)	4.04 (0.33)	3.79 (0.06)	3.62 (0.09)	5.62 (0.11)	3.88 (0.03)
MMIFR (liters/s)	4.50 (1.41)	1.88 (0.65)	4.21 (0.38)	8.00 (0.19)	3.42 (1.10)	1.91 (0.04)	4.04 (0.48)	5.04 (2.51)	4.78 (0.90)	1.32 (0.23)	3.12 (0.49)	5.34 (1.75)	3.16 (0.45)	1.39 (0.09)	3.01 (0.93)	5.70 (0.10)

* Abbreviations as in Table 2.

**N = 4

TABLE IV. Conditions for Acceleration Runs

Independent Variables*	NO M-1 Maneuver		M-1 Maneuver	
	SBA = 15°	SBA = 75°	SBA = 15°	SBA = 75°
1. Plateau G level (G)	3 & 4	4 & 5.5	4 & 5	5 & 7
2. Plateau G duration (s)	20, 40	20, 40	20, 40	20, 40
3. Interpulse period (s)	4, 8, 32	4, 8, 32	4, 8, 32	4, 8, 32
4. AGS inlet press. (psig)	2.25 & 3.75	2.00 & 3.20	3.75 & 5.25	2.80 & 4.40

*All levels of variables 2 and 3 were used with each combination of variables 3 and 4. Only corresponding levels of variables 1 and 4 were used in any particular run.

TABLE V. Pulmonary Function Measures Obtained by Use
of the Portable Respirometer (Phase III).
(Entries are mean, standard deviation, and N.)

Subj	SBA [†]	FVC ^{††} (liters)	FEV ₁ (liters)	FEV ₃ (liters)	MMEFR (liters/s)	FIVC (liters)	MMIFR (liters/s)
1	15°	3.80 ^{***} 0.30 8	2.63 ^{***} 0.17 8	3.44 ^{***} 0.21 8	1.81 0.22 8	3.29 [*] 0.25 8	4.74 0.57 8
	75°	3.22 0.16 6	2.24 0.12 6	2.95 0.16 6	1.61 0.21 6	2.98 0.20 6	4.78 0.90 6
2	15°	4.24 0.65 7	1.70 0.92 6	3.45 0.75 6	1.32 0.53 6	2.96 0.29 6	1.38 0.25 6
	75°	—	—	—	—	—	—
3	15°	5.70 ^{**} 0.37 8	4.24 ^{**} 0.34 8	5.37 ^{**} 0.27 8	4.13 [*] 0.52 8	5.33 ^{***} 0.37 8	2.71 0.62 8
	75°	4.93 0.45 6	3.69 0.25 6	4.72 0.38 6	3.52 0.37 6	4.51 0.28 6	2.33 0.26 6
4	15°	3.84 ^{**} 0.25 7	3.12 ^{**} 0.24 7	3.67 0.21 7	4.11 ^{***} 0.54 7	3.56 ^{**} 0.26 7	6.06 [*] 1.58 7
	75°	3.20 0.32 6	1.56 0.96 6	2.93 0.23 6	2.63 0.37 6	3.01 0.25 6	4.32 0.35 6

† SBA = Seat Back Angle

†† Abbreviations as in Table 2. Asterisks in SBA = 15° entry blocks show t-test probabilities of corresponding 15° and 75° means, as follows: *p = 0.05; **p = 0.01; ***p = 0.001.

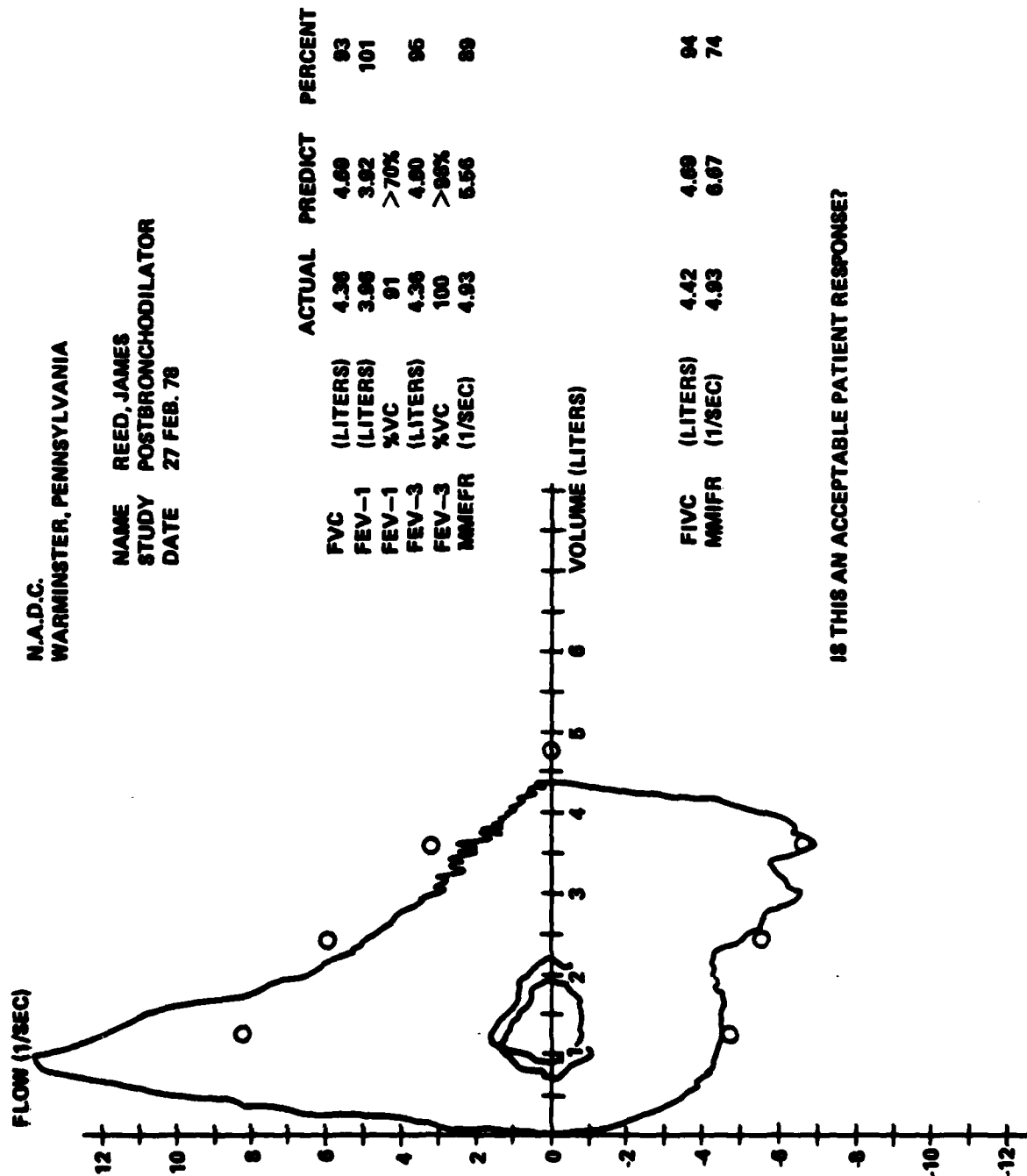


Figure 1. Example of Flow-Volume Loop and automated analysis

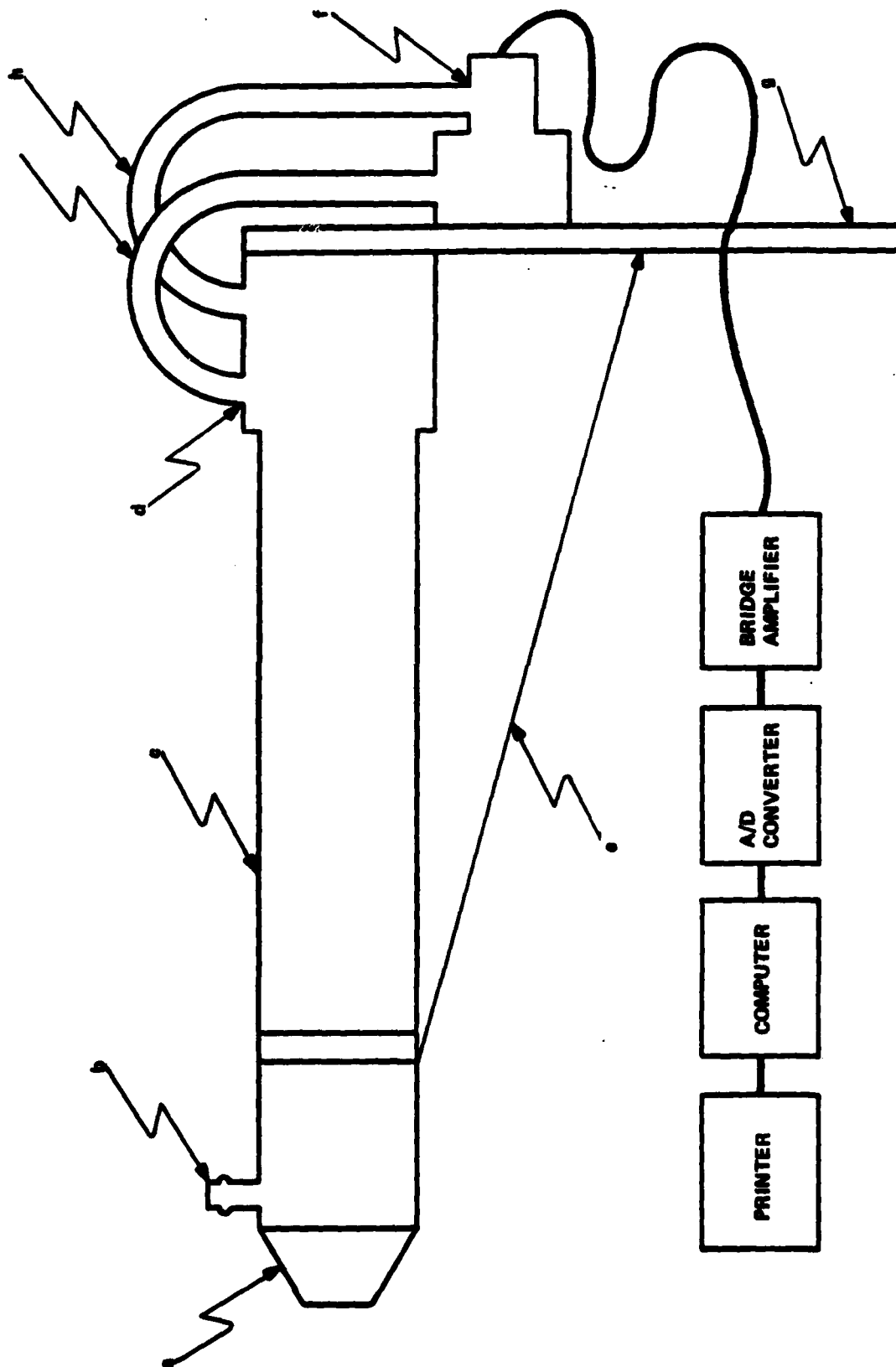


Figure 2 — Diagram of portable respirometer: a. mouthpiece, b. tap (mouth pressure), c. metal tube, d. pneumotachograph, e. supporting fin, f. pressure transducer, g. mounting plate, h. flexible tubing connecting pneumotachograph to pressure transducer.

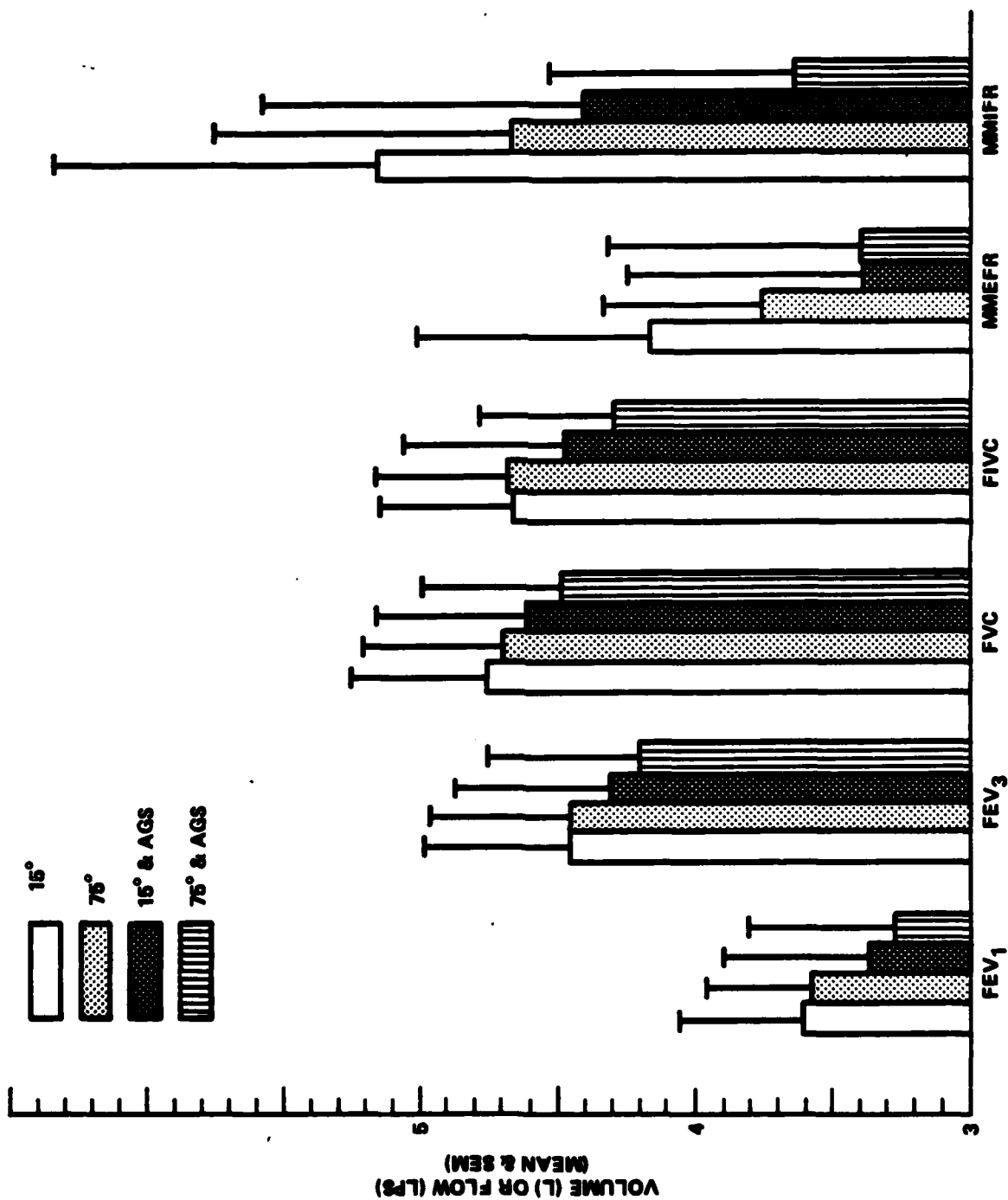


Figure 3. Mean maximum values of pulmonary function measures from baseline FVC testing (Phase II)

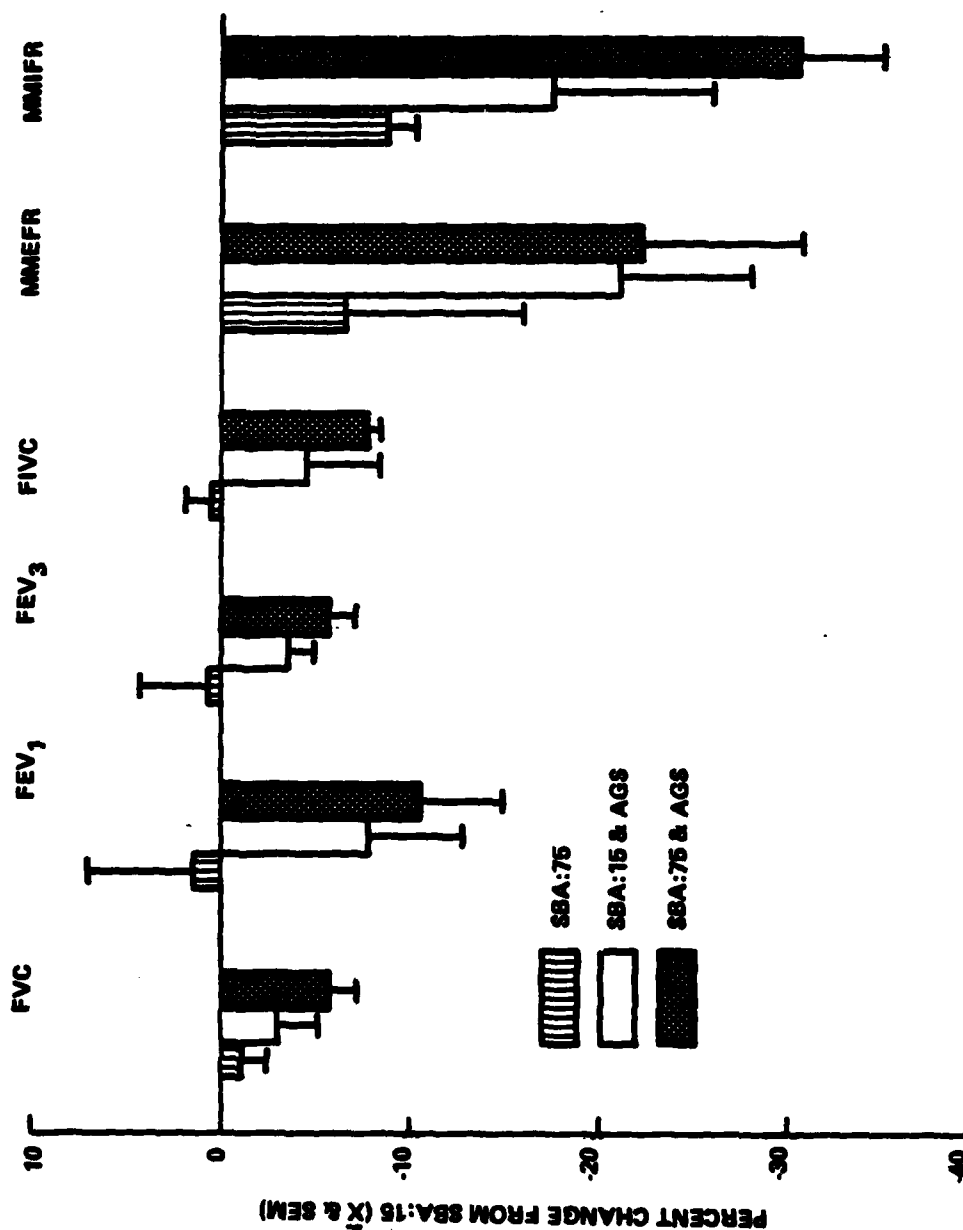


Figure 4. Percent changes (mean and standard error of the mean) in mean maximum values of FVC testing measures relative to those measured at SBA = 15° (Phase II)

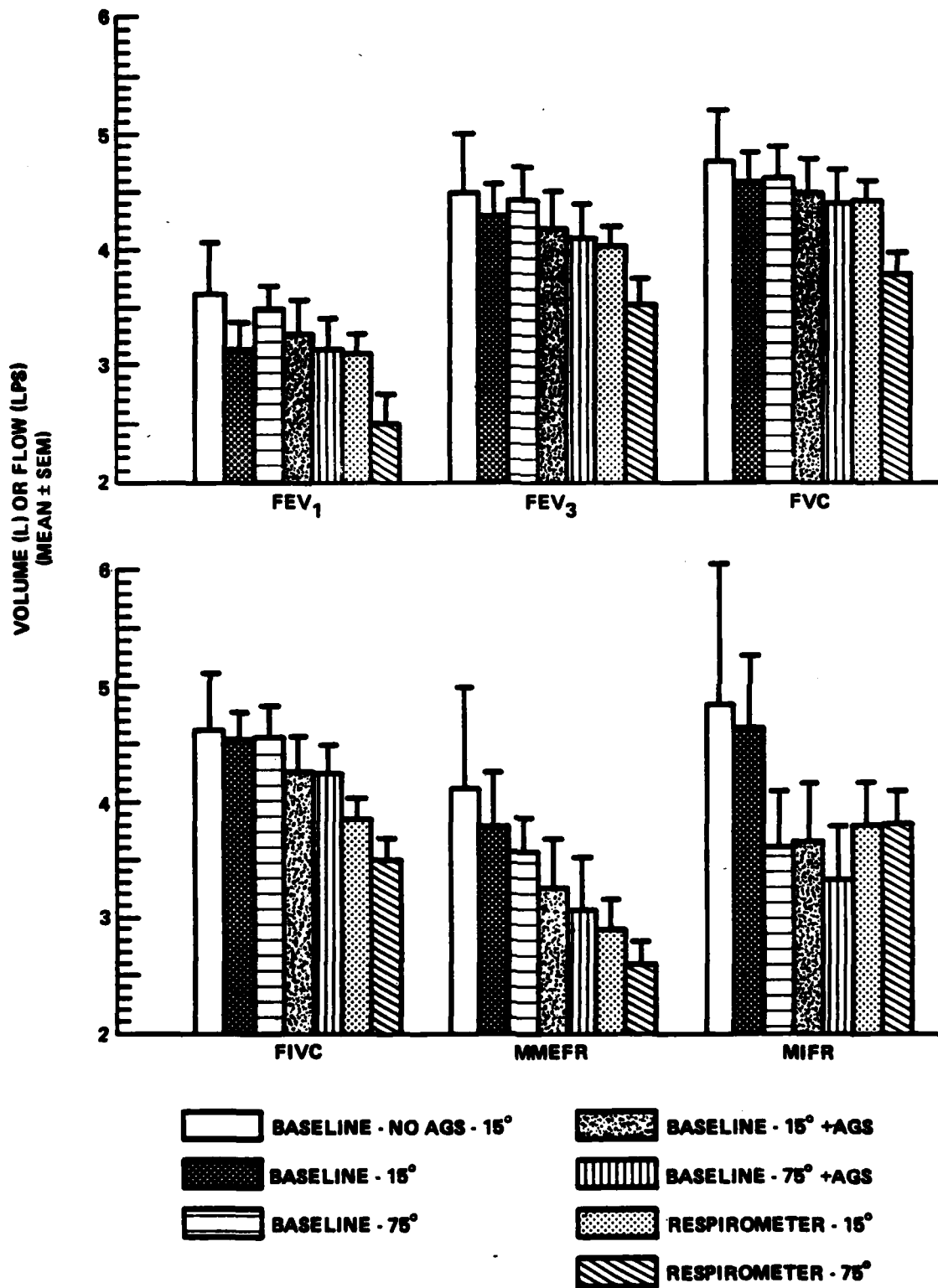


Figure 5. Mean and standard error of the mean for mean values of pulmonary function measures in Phases I, II, and III

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