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BREATHING RESISTANCE AND DEAD SPACE IN RESPIRATORY PROTECTIVE DEVICES

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16. Abstracts A review reveals that respirators, in general, are worn by only about 20% of workers in need of respiratory protection against hazardous substances in the workplace, primarily due to generalized "discomfort". Breathing resistance and equipment dead space are two of the most important design factors contributing to this discomfort. The principal physiological responses to added breathing resistance appear to be hypoventilation, reduced oxygen consumption, a "flattened" and prolonged pattern in the breathing phase to which resistance has been added, increased respiratory work, and a tendency for increased functional residual lung capacity and increased carbon dioxide retention. Added dead space forces rebreathing of exhaled carbon dioxide which, in turn, stimulates compensatory hyperventilation to maintain a normal alveolar PCO_2. The mechanism for maintaining a normal PCO_2 seems effective up to 2% for short-term and to 1% CO_2 for long-term exposures.

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BREATHING RESISTANCE AND DEAD SPACE IN RESPIRATORY PROTECTIVE DEVICES

Physiological Effects of Breathing Resistance and Equipment Dead Space in Respiratory Protective Devices: Status of the Problem

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ABSTRACT

Respirators, in general, are worn by only about 20% of workers in need of respiratory protection against hazardous substances in the workplace. This has been principally due to a generalized "discomfort" experienced by workers. Breathing resistance and equipment dead space are two of the most important design factors contributing to this discomfort.

The principal physiological responses to added breathing resistance appear to be hypoventilation, reduced oxygen consumption, a "flattened" and prolonged pattern in the breathing phase to which resistance has been added, increased respiratory work, and a tendency for increased Functional Residual Lung Capacity and increased carbon dioxide retention (when compensation is incomplete).

Added dead space forces rebreathing of exhaled carbon dioxide which, in turn, stimulates compensatory hyperventilation to maintain a normal alveolar PCO_2. Tidal volume increases and, thereafter, breath frequency rises. The mechanism for maintaining a normal PCO_2 seems effective up to 2% CO_2 for short-term and to 1% CO_2 for long-term exposures.

Additional studies of physiological responses under stress and reconsideration of current standards are recommended.

INTRODUCTION

Respiratory protective devices have been in use for many years, at work sites where sufficient engineering or work practice controls have not been possible or feasible. Some regulations governing their use and performance have been in existence since 1919, with the latest and most comprehensive requirements given in the Federal Register, March 25, 1972.[1] The Bureau of Mines first established requirements under schedule 21 in 1934.[2]

Although these regulations were designed to ensure that maximum protection be provided for individual workers under a variety of conditions, investigations have shown that respirators, in general, are only worn by a low percentage of those in need of respiratory protection (approximately 20% in three studies).[3,4,5] This has been principally due to a generalized discomfort experienced by workers. Frequently, they are worn only intermittently, mainly when air contamination is severe. The effectiveness of respirators in reducing work-related illness is thus thwarted by lack of use, which is to a large extent due to design factors in need of improvement. In addition, on-site investigations as well as laboratory sampling studies have shown that a significant face-mask leakage problem exists, especially in the quarter and half mask designs.[4,6,7,8]

The discomfort found in wearing respirators has been found to be due, principally, to the following effects: (1) pressure on the face (2) high breathing resistance (3) interference with heat loss and (4) fog on glasses.[9] Interference with vision has been found and assessed quantitatively.[10]

It has also been shown that large equipment dead space in full face masks and in some half masks will cause inhalation of expired carbon dioxide in concentrations sufficient to produce involuntary hyperventilation in response to the CO_2.[11,12,13,14,15] This will also significantly contribute to the sense of discomfort encountered with respirator use. In addition, there will be a decrease in the efficiency of lung ventilation, while compensating for the added dead space.[10] Furthermore, an inability to adequately compensate (through hyperventilation) may produce respiratory acidosis in workers with abnormalities of pulmonary ventilatory function.[16]

Although many studies have been done in an effort to determine acceptable levels of resistance in breathing apparatus, levels at which no significant acute or long-term adverse physiological effects occur have not been defined. The present upper allowable limits seem to have been based principally on studies by Silverman[17,18,19,20] Cooper[21,22] and Senneck.[23]

A brief review of findings of past investigations dealing with effects of added resistance to air flow and added anatomical (instrumental) dead space encountered in wearing respirators follows.

EFFECTS OF ADDED BREATHING RESISTANCE

Early studies on breathing resistance dealt principally with problems encountered in gas masks, especially those used during World Wars I and II.

Prior to 1945, there was little quantitative data on the effects of moderate resistance on respiration and circulation. The conclusions of workers were essentially (as summarized by Cooper, 1960)[21] that resistance causes a reduction in minute volume, usually through a reduction in breath frequency; a tendency toward carbon dioxide (CO_2) retention and reduced oxygen uptake (establishing an oxygen debt); and a fall in cardiac output. The effects of expiratory resistance are greater than inspiratory. Responses were diminished by deep inspiration and by training in breathing against resistance to air flow.

STUDIES TO 1965

Silverman (1945)[17] studied normal young men who worked on a bicycle ergometer at 415, 830, and 1,107 kg-m/min with inspiratory resistances from 6 to 106 mm H_2O (measured at 85 L/min air flow, established as the maximum flow to be encountered under work conditions, assuming maximum flow to be twice expiratory minute ventilation). The responses suggested that most subjects could tolerate these resistances unless the total external respiratory work exceeded 2.5, 6.0, and 13.3 kg-m/min, respectively. They recommended that external respiratory work should not be greater than 0.6% of total body work. They also observed that: (1) added resistance reduces expired minute volume ($\dot{V}_E$) and breath frequency, but physiological mechanisms could compensate for effects (2) if expiratory resistance was greater than inspiratory, the oxygen uptake ($\dot{V}O_2$) was reduced (3) if more inspiratory resistance was added, $\dot{V}O_2$ tended to rise (4) subjective discomfort was noted if expiratory resistance was greater than the inspiratory and both were greater than 50 mm H_2O at 85 L/min and (5) training reduced the effects of added resistance. Silverman (1946)[24] later found added resistance to cause a smoothing and damping of the air flow curve.

The same changes in flow characteristics were found with external resistance to air flow in a study by Cain and Otis (1949).[25] Elimination of the pause before start of inspiration was also noted when resistance was added. Tidal volume, minute volume, and frequency changes were similar to those found by Silverman. All subjects showed increased $PACO_2$* and decreased PAO_2** with added resistance. Pressure-volume plots demonstrated the increased work of breathing.

In a later study of the effects of resistance on air flow by Silverman et al. (1951),[18] which was designed to cover a wider range of work rates (to 1660 kg-m/min), comparisons were made with resistance of 64 mm H_2O inspiratory and 41 mm H_2O expiratory (considered comparable to war gas masks); 6 mm H_2O inspiratory and 3 mm H_2O expiratory (minimal resistance). They found the effects of resistance similar to that previously reported (1945).[17] At 830 kg-m/min the added resistance (64/41) significantly lowers $\dot{V}O_2$ as compared to the minimal resistance condition at that level. Tidal volume and respiratory quotient (RQ) were also significantly raised. A separate experiment with 64 mm H_2O inspiratory and 27 mm H_2O expiratory resistance at 415 kg-m/min showed no significant difference as compared to the 64 mm H_2O/41 mm H_2O resistance at this level.

The reduction in minute volume due to added resistance was confirmed by Morrow (1952-53)[26] and others. McIlroy (1956)[27] reported that the resistance to air flow caused a reduction in breath frequency and a rise in respiratory level corresponding to a larger Functional Residual Capacity (FRC), whereas elastic resistance (resistance to elastic stretching or recoil of lungs and thorax) resulted in increased frequency of breathing and a fall in respiratory level (FRC). Campbell (1957)[28] also found that resistance to expiratory air flow causes a rise in FRC.

Cooper (1960)[21] and Senneck (1962)[23] reviewed the data of Silverman et al., and derived standards for the maximum allowable total respiratory work in a breathing apparatus, based upon a more accurate description of respiratory wave form. The recommendation for maximal respiratory work then becomes 0.74% of the total body work. Cooper (1960)[21] also criticized Silverman's recommendation that 64 mm H_2O (inspiratory)/41 mm H_2O (expiratory) be the maximum allowable resistance at 1107 kg-m/min. Although he observed that resistances of 82/53 could

* partial pressure exerted by carbon dioxide in the alveolar gas
** partial pressure exerted by oxygen in the alveolar gas

be borne by men at work levels of 830 kg-m/min, he concluded that the standard might not be strict enough. The subjects were not free of discomfort and some adverse physiological effects ($\dot{V}E$, $\dot{V}O_2$, and CO_2 output fell) were found at high levels of exertion. Also most experiments lasted only 15 minutes.

Cooper (1960)[21] also stated that added resistance is undesirable in two ways: (1) the respiratory muscles may not be able to do the work necessary to ventilate the lungs (2) the resistance combination between the two breathing phases may alter the fundamental relationship between ventilation and circulation, adversely affecting gas transfer.

Silverman and Billings (1961)[20] maintained that the maximum inspiratory air flow should be four times the minute volume, if the shape of the flow curve approximates a triangle rather than a rectangle, which had been the assumption for selection of 85 L/min as the test flow level (based upon twice an inspired minute volume of 42.5 L/min obtained during a slow run and that inspiration represents half the respiration cycle). They maintained, therefore, that the Bureau of Mines, 1955 A and B schedules, did not provide a satisfactory basis to evaluate protection. The Bureau of Mines requirement at that time (inspiratory resistance no greater than 89 mm H_2O in gas masks and 50 mm H_2O in dust respirators, at 85 L/min flow; with expiratory resistance not to exceed 38 mm H_2O and 28 mm H_2O, respectively) seemed to have an arbitrary basis (Silverman, 1961).[20] They were selected principally on the manufacturer's ability to meet the specification. Silverman's data showed that (1) the respiratory system adjusts readily (within limits) to changes in inspiratory or expiratory resistance (2) the phase of the cycle in which resistance is present becomes prolonged and maximum flow is reduced for the same minute volume (3) the phase of the cycle without added resistance manifests higher flows and less time for completion (4) oxygen deficit increased in proportion to total resistance, and (5) the effect of expiratory resistance is much greater than inspiratory. Subjective reactions, which were used as a basis for approval or disapproval of respirators prior to that time, were found to correlate very poorly with objective data. However, a greater frequency of undesirable responses was obtained when the total resistance was highest and the reactions were found in proportion to resistance when the resistance exceeded 64 mm H_2O (inspiratory) and 41 mm H_2O (expiratory). His data showed that most physiological reactions to resistance are not a function of the total resistance but rather a function of the ratio (balance) between the inspiratory and expiratory resistance. Oxygen debt seemed to occur when expiratory resistance was greater than inspiratory. He also concluded that inspiratory resistance above 82 mm H_2O and expiratory resistance above 53 mm H_2O are not desirable, based upon a high percentage of unfavorable responses and on the fact that significant changes in the physiological variables studied were observed below those values. The mean data showed that the increased oxygen debt observed was a function of total resistance (inspiratory plus expiratory). Pulse rate was not significantly altered by added resistance.

Muira and Kimura (1960)[29] also found a reduction in respiratory rate (most conspicuous effect) and a high CO_2 tension in the blood. They concluded that the desirable limit of maximal inspiratory resistance during moderate muscular work should be about 60 mm H_2O at 100-130 L/min air flow. This was based upon tests using inspiratory resistances of 1, 4.5, and 10 mm H_2O at 30 L/min flow with lower expiratory resistances, which corresponded to inspiratory resistances of 6, 21, and 55 mm H_2O at 85 L/min, respectively, and to 9, 42, and 90 mm H_2O at 120 L/min, respectively.

McIlroy et al. (1956)[30] reported on his adaptive studies of mechanisms in man when subjected to both added elastic (5.6 cm H_2O/L/sec) and nonelastic (13 cm H_2O at 60 L/min) resistances. They observed a reduction in breath frequency by an external resistance even at rest, with a simultaneous increase in tidal volume, to maintain the appropriate alveolar ventilation. Their data showed that compensatory response to added resistance can be accomplished either by the respiratory rate or the respiratory level being altered, with a combination of the two occurring in most cases (interdependency). Breathing is adjusted so that a given ventilatory task is accomplished with a minimum of respiratory effort.

The maximum breathing capacity can also be significantly lowered with added resistance to breathing above 5.2 cm H_2O at 3.8 L/sec and 2.0 cm H_2O at 1.5 L/sec as shown by Zwi et al. (1959)[31]. There is a much greater reduction in patients with obstructive airway disease than in normals.

STUDIES SINCE 1965

The effects on mechanics of breathing were observed by Hanson et al. (1965).[32] A significant decrease in pulmonary compliance with increased nonelastic work was found, using an expiratory resistance corresponding to 55 mm H_2O at 45 L/min flow. These authors had previously, with B.S. Tabakin (1965),[33] discovered significant changes in residual volume (RV), expiratory reserve volume (ERV), and the lung clearance index (LCI), which is a measure of distribution of inspired air. Modification of gas exchange mechanisms in the presence of external expiratory obstruction had been previously found by these same investigators working with A.M. Levy (1961).[34]

An examination by Thompson and Sharkey (1966)[35] of the recovery $\dot{V}O_2$ after exercise with half masks and full face masks, which produced anaerobic metabolism due to excessive breathing resistance, resulted in their finding that the resistance to breathing and the recovery O_2 consumption values were positively related at the higher work loads. There were significant differences in recovery O_2 consumption values at light, moderate, and heavy work loads. Subjective responses supported the conclusion that there was excessive resistance in the masks.

Gee et al. (1968)[36] studied the effect of inspiratory resistance alone, expiratory resistance alone, and combined resistances on intra-pulmonary gas exchange and $\dot{V}O_2$ up to near $\dot{V}O_2$ max.* They found: (1) mean mouth pressure approximately 2/3 of peak pressure (2) progressive hypoventilation occurred as work load increased in all cases (3) tidal volume decreased significantly with increased expiratory obstruction, but not with inspiratory and combined obstruction (4) breath frequency decreased with inspiratory obstruction and combined obstruction but not with expiratory obstruction (5) no significant changes in $\dot{V}O_2$ occurred (6) the magnitude of reciprocal changes in estimated $PACO_2$ and PAO_2 increased with the work level (7) diffusing capacity for carbon monoxide (DLCO) was not affected and (8) heart rate was not affected. They used a 5 cm H_2O/L/sec at 2 L/sec air flow resistance at 800, 1200, and 1600 kg-m/min work levels. They concluded that the data showed no major changes in intra-pulmonary gas exchange and that this degree of obstruction does not limit work capacity for everyday tasks, based upon $\dot{V}O_2$, heart rate, and absence of subjective discomfort.

Cerretelli et al. (1970)[37] found decreased $\dot{V}O_2$ and $\dot{V}E$ as the resistance was increased at any exertion level, but that the relationship between $\dot{V}O_2$ and work load remains unchanged. They stated that there was no indication that increased external resistance placed on the ventilatory apparatus cause a shift to anaerobic metabolism. Rather, the reduction in work output seems to intervene prior to development of a detectable oxygen deficit.

Craig et al. (1970)[38] suggested that the minimum time for expiration may be a useful index to determine when limiting resistance to breathing is encountered at any work level. In their study, using inspiratory resistances of from 1.5-15.5 cm H_2O/L/sec (15-155 mm H_2O at 60 L/min) and expiratory resistance

* maximum aerobic oxygen consumption

of from 2.0-3.9 cm H_2O/L/sec (20-39 mm H_2O at 60 L/min), they found little effect on endurance, no significant change in $\dot{V}O_2$ (with high inspiratory resistance) and greater endurance in the presence of CO_2 concentrations up to 3%.

Freedman and Campbell (1970)[39] tried to determine the ability of normal subjects to tolerate added inspiratory mechanical loads. They found the maximum tolerable levels were reproducible and in 10 subjects were:

elastic (effect of a decreased thoracic compliance) 145 cm H_2O/L

viscous (flow resistive) 283 cm H_2O/L/sec

threshold (pressure developed before flow can occur) 75 cm H_2O.

They made reference to Mead's finding (1966)[40] that, at rest, a change from mouth breathing to breathing through the nose is accompanied by a doubling of the work of breathing.

On the basis of new "self-rescuer" filter-type breathing apparatus in the British coal industry, Bentley et al. (1972)[41] re-evaluated the resistance problem, using graded resistance levels comparable to that actually encountered in practice, from the standpoint of subjective response, i.e., ability to tolerate the added respiratory load (no general physiological relationship implied). Only inspiratory resistance was added. A highly significant correlation was found between the proportion of subjects noting discomfort and the additional respiratory work done per liter of air inhaled (kg-m/L) and versus the peak inspiratory pressure swing. The degree of dyspnea was, therefore, a function of negative intrathoracic pressure. They noted that the resistance levels investigated (similar to those evaluated by Silverman, 1945) were between those detectable but not uncomfortable and those that are maximally tolerable. The "compromise" standard suggested was set at 0.14 kg-m/L of air inhaled so that 90% of the population would not experience respiratory discomfort. That value was given as equivalent to a pressure drop across the inspiratory valve of 14 cm H_2O during conditions of steady flow. Ten percent of the population experienced some discomfort when the peak inspiratory pressure exceeded 19.5 cm H_2O. It was suggested, therefore, that the peak pressure drop across the breathing equipment should not exceed 22.3 cm H_2O, when testing with a sine wave pump, at flow levels attainable by normal men during sustained maximal exercise for 30 minutes.

Another report of the same study of subjective response (Bentley et al. 1973)[42] stated that peak inspiratory pressure and inspiratory work rate per liter of air inhaled are good predictors of discomfort but that inspiratory work alone is not. This finding supported the conclusions of Silverman and Cooper, but not McIlroy, who considered absolute respiratory work as the major factor in developing dyspnea. There was considerable variability in the discomfort felt by different men under similar conditions of breathing. They also found peak flow to approximately equal 2.7 times the minute volume.

T. Comte (1971)[43] reported on a study comparing three types of respiratory apparatus (two gas masks and a unit for sand blasting) which included measurements of blood pH and PCO_2. They found physiological disturbances in all indices at different work levels (230-600 kg-m/min). The responses were attributed to excessively large dead space in the mask (about 380 ml), excessive inspiratory and expiratory resistance, and insufficient volume of air. The blood pH did not decrease beyond 7.35 and PCO_2 did not increase beyond 45 mm Hg, though. This author considered 29 mm H_2O as too high for acceptable inspiratory resistance. She stated that it was necessary to reduce the respiratory load by modifications to reduce both resistances and mask dead space, secure adequate air changes, and reduce weight.

Martin et al. (1971)[44] studied the distribution of ventilation as affected by respiratory effort in four subjects. He concluded that variations in respiratory effort do not produce variations in dynamic pleural pressure between lung regions during either inspiration or expiration.

L. Hermansen et al. (1972)[45] compared exercise tests with a gas mask versus a conventional respiratory valve. The resistances encountered were: inspiratory 9 cm H_2O/L/sec (90 mm H_2O at 60 L/min), and expiratory resistance 2.6 mm H_2O/L/sec (26 mm H_2O at 60 L/min), versus inspiratory 1.7 cm H_2O/L/sec (17 mm H_2O at 60 L/min), and expiratory 1.7 cm H_2O/L/sec (17 mm H_2O at 60 L/min), respectively. There was no difference in $\dot{V}O_2$ until the 1200 Kpm work level was reached (approximately 75% of maximal work capacity). $\dot{V}O_2$ max decreased significantly during maximal exercise with the mask. $\dot{V}_E$ was always lower with the mask, and the difference became greater with increased work load. Breath frequency also drops with the mask but was partly compensated for by increased tidal volume. This ventilatory compensation failed at $\dot{V}O_2$ max. Heart rate was higher with the mask at submaximal exercise. Their findings supported other authors in concluding that there is an impairment of work capacity by external resistance to breathing. Hermansen noted that pulmonary ventilation is not believed to be the limiting factor for maximum performance in healthy young subjects. $\dot{V}O_2$ max will drop, however, when ventilation is hampered.

L. Chretien et al. (1973)[46] stated that the limit for additional work of breathing imposed by wearing a protective respiratory mask should agree with the recommendations of Cooper (1960) (restricting energy in watts to 2% of the ventilation value). A method was developed for measuring this respiratory work. From their study, they concluded that filtering devices are acceptable for moderate work (<90 watts), self-contained closed circuit devices make vigorous activities possible for several hours, and self-contained open circuit devices are best for short, extreme efforts (20 to 30 min).

T. Comte et al. (1975)[47] also tested subject reactions to four different respirators. They found that at 50% $\dot{V}O_2$ max there was an excessive load on the physiological functions of the organism. They concluded that air flow resistance should not exceed 33 mm H_2O during light work (30% $\dot{V}O_2$ max) or 20 mm H_2O during moderate work (50% $\dot{V}O_2$ max). They also recommended a work/rest regimen for workers.

The ability of man to detect changes in his breathing was examined in four normal subjects by West et al. (1975),[48] using a raised PCO_2 to increase ventilation. Detection occurred when tidal volume increased from normal values by an average of 700 ml (550-890 range) among the subjects, with the detection level being constant for each individual.

Johnson (1975)[49] discussed development of the ratio of inhalation time to exhalation time during the respiratory cycle as an index of prediction of exercise tolerance time, based upon the concept of optimization of respiratory characteristics (minimum work performed) as limited by respiratory stress. Exhalation time as an index of the voluntary endpoint of exercise was studied earlier by this author (Johnson, 1974).[50] He found this to be a rapid and reliable measure of the state of respiratory exhaustion. The average minimum exhalation time in 18 healthy, young male subjects was 0.66 sec. The effect of exhalation time on exercise performance was also emphasized by this author in a later paper.[51]

In a recent study (Woodcock et al., 1976)[52] the detection of differences in breathing resistance between powered (positive pressure, continuous flow) air purifying respirators and conventional respirators was examined. It was concluded that subjective judgement of external breathing resistance is sensitive and consistent. The conventional respirator had 50 mm H_2O inspiratory and 12 mm H_2O expiratory resistance (at 85 L/min). The powered respirator delivered 3.2 cfm. Comparisons were made at 400 and 580 kg-m/min. The change in inhalation resistance could be detected at both work levels.

In another new paper (Johnson, 1976),[13] the energy requirements of respiration were discussed. The addition of a mask adds to the disproportionate increase in respiratory energy requirements as the overall body energy expenditure increases with exercise. He stated that masks have been improved considerably, but still contribute resistance and dead spaces approximately equal to those normally found in the human body. He also referred again to the minimization of respiratory effort in adapting to increases in resistances and dead space encountered in masks. Resistance of the U.S. Army M17 mask were given (from Comroe, 1965)[53] as: inhalation, 3.4 cm H_2O/L/sec, and exhalation, 1.3 cm H_2O/L/sec. The values given for an average normal human without mask were: inhalation, 1.6 cm H_2O/L/sec, and exhalation, 1.7 cm H_2O/L/sec. This, in combination with the increase in mask dead space (350 ml, M17 Army mask versus average normal human dead space of 150 ml) causes a sudden onset of respiratory disturbance to the respiratory center. The major problem is that the air flow resistance of the mask increases with exercise (dependent upon valves and other characteristics of the mask) in a non linear relationship. He states that the respiratory work increases from 8-10% of the body $\dot{V}O_2$ during severe exercise to probably 15% or more when the mask is added. Compensatory adjustments are made by the body as exercise progresses to improve efficiency of the respiratory system (e.g., opening of airways). But, while body airway resistance to flow is decreasing, the resistance to air flow through most masks increases. He states that, "In terms of energy requirement, the mask is very burdensome indeed."

EFFECTS OF ADDED DEAD SPACE

The problem of response to rebreathed CO_2 in a respirator mask was not dealt with as early as was the problem of resistance to breathing. The concern about CO_2 concentrations inhaled with each breath seems to have developed principally from NASA studies.

Specifications for maximum allowable concentrations were set, however, by the Bureau of Mines prior to 1966. During human testing, the average CO_2 concentraton was not to exceed 2.0% in the wearer's breathing zone.[54] Kloos and Lamonica (1966)[54] developed a machine test method because they claimed that breath-to-breath variations prevented accurate evaluation.

The 1977 specifications unchanged from those listed in the Federal Register, March 26, 1972[1] sets limits dependent upon exposure time: 2.5% for 30 minutes of less, 2.0% for one hour, 1.5% for two hours, 1.0% for three hours, 1.0% for four hours. For a closed circuit apparatus, only 0.5% is permitted.

The basic physiological response to inhaled CO_2 is that over a considerable range, V_E increases directly with CO_2 concentration, with an initial increase in tidal volume. The high CO_2 concentration raises the arterial PCO_2 which stimulates faster and deeper breathing. This is a compensatory mechanism, which is effective up to about 2.0 volume-percent. Further increases in CO_2 concentration, dependent upon time as well, "produce symptoms such as discomfort, fatigue, dizziness, headache, ringing in the ears, drowsiness, paralysis of the respiratory center, and finally asphyxiation and death" (Kloos and Lamonica, 1966).[54] Even at 2% CO_2, there may be discomfort when the apparatus is worn for two hours or longer (Kloos and Lamonica, 1966).[54] The TLV for an eight hour workday is 0.5% CO_2 (5000 ppm). Kloos and Lamonica[54] also stated that modern respirator facepieces still "have considerably increased dead space." It is the effective dead space, however, which determines the actual inhaled CO_2 concentration, due to some air around the edges of the facepiece being almost static and the fact that nose cups used to prevent fogging in full facepieces decrease effective dead space, and thereby inspired CO_2 (Kloos and Lamonica, 1966),[54] (Christman et al. 1974).[10] The adequacy of closed circuit apparatus depends upon the effectiveness of the CO_2 absorber used in each case.

An early study (Samet et al.)[16] showed that, although moderate to severe obstructive airway disease can cause, in the presence of increased inspired CO_2, an increase in arterial PCO_2 ($PaCO_2$) and decrease in pH, mild degrees of obstruction have relatively little effect upon the response to inhaled CO_2. They also stated that ventilatory response to inhaled CO_2 may be impaired in normal subjects by an extended stay in a high CO_2 environment.

In a NASA-sponsored study by the Lovelace Foundation for Medical Research (1971),[12] a highly significant reduction in $\dot{V}O_2$ max (13%) was found when inspired gas contained 15 mm Hg PCO_2. There was also CO_2 retention and acidosis with this CO_2 level at the endpoint of exercise and during recovery. They claimed that inspired CO_2 at that concentration "adds to the metabolic energy requirement and taxes the life support system." At 9.4 mm Hg PCO_2, there was only a moderate increase in ventilation and some evidence of inhibition of CO_2 elimination (not statistically significant). The effects of inhaled CO_2 were decreased with increased exercise levels. They also found the increases in ventilation roughly proportionate to PCO_2 inhaled (log ventilation versus metabolic rate as a fraction of $\dot{V}O_2$ max). Under basal conditions and inspired PCO_2 of 7.5 mm Hg. all subjects increased their ventilation (19%). There also was a significant increase in alveolar PCO_2 (1.5 mm Hg) and in PO_2 (4.7 mm Hg).

Jones et al. (1971)[55] determined that the resulting increases in ventilation and alveolar PCO_2 were dependent on the work load and volume of added dead space. In their study, the added volumes were 700 and 1400 ml. They found no consistent effect on CO_2 output or O_2 uptake. Ventilation increased progressively, due principally to an increased tidal volume. The increased ventilation was insufficient to restore the alveolar PCO_2 to normal even though the ventilation levels were usually within the subject's capability.

In a study by Zagryadskiy (1971),[56] when the rate of CO_2 accumulation decreased, the subject was better able to compensate and tolerate an increase of up to 6% CO_2. He also noted that $PACO_2$ increases only 2 to 3 mm Hg at 2.5 - 3.0% CO_2. The initial reaction to inhaled CO_2 was also found to be an increased tidal volume.

Continuous monitoring of expiratory flow, PO_2, and PCO_2 to yield breath-by-breath values, while breathing 3, 5, 6 and 7% CO_2 (Reynolds et al. 1972)[57] showed $\dot{V}_E$ increasing smoothly at all stimulus levels, tidal volume changing rapidly and stable thereafter, while breath frequency increased much more slowly. PAO_2 changed rapidly under stimulus and slowly after termination of the CO_2 stimulus. $PACO_2$ responded faster both on-stimulus and off-stimulus.

Specific airway conductance (GAW) decreased significantly in nine normal subjects while alveolar PCO_2 increased 5.0, 12.3, and 20.2 mm Hg with breathing of 5, 7.5, and 10% CO_2, respectively (Tashkin and Simmons, 1972).[58] They concluded that normal airways constrict in response to mild to severe hypercapnia.

The hyperventilation caused by CO_2 inhalation was only detectable subjectively when a group mean tidal volume increased by 700 ml (West et al. 1975).[48]

Approximately double the ventilation/max ventilation ratio occurred at three levels of exercise when dead space of 1200 ml was added (Kelman and Watson, 1973).[59] There were no significant increases in heart rate or VO_2.

Hey et al. (1966)[60] and Cotes et al. (1970)[61] showed that $\dot{V}_E$ increases under progressive exercise conditions, by the tidal volume increasing to about 50% of the VC and then further by increased breathing frequency, under normal conditions. Kelman and Watson showed, however, that the limit to the tidal volume/vital capacity ratio increase is nearer to 70% when the respiratory dead space is artifically increased.[59,62]

A re-breathing method for testing sensitivity to CO_2 was described by Milic-Emili (1975).[63] This was considered important because of the current argument that persons with an initial low sensitivity to CO_2 are those in whom CO_2 retention occurs first when airway obstruction develops.

Stanley et al. (1975)[64] stated that there is an inhibitory effect of lung inflation on respiratory drive in man. Breath-holding time is longer at high lung volumes than at low volumes.

The concentration of inhaled CO_2 may be estimated from knowledge of respirator mask dead space and average tidal volume at each work level. Based upon the assumption that effective mask dead space varies from 100 to 250 ml (Hermansen et al. 1972 ;[45] Johnson, 1976 ,[13])some hyperventilation would be expected in at least some cases, dependent upon exposure time. The findings of Johnson and Cummings (1975),[65] that some effect is expected above 100 ml dead space support this expectation. Approximately 1% (7.6 mm Hg) CO_2 has also been established as the acceptable limit (Michel et al. 1969)[66] in a protective suit. The prediction formula of Johnson and Cummings (1965)[65] ($\Delta\dot{V}_E$(L/min)= $12\Delta V_d$(L)* can be used to estimate the degree of response. Large mask dead spaces appear to be common (when secured onto a standard male head manequin, a sample Wilson half mask contains 260 ml total dead space and an MSA full face mask contains 805 ml total dead space). Up to 190 ml dead space was found in measurements of 15 different dust respirators examined in France.[10] The U.S. Army M17 mask has 350 ml dead space (Johnson, 1976).[13] Some CO_2 response is, therefore, expected at exercise levels, as well as at rest, although decreased as tidal volume increases with exercise.

SUMMARY

The effects of added resistance, in general, may be summarized as follows:

> Hypoventilation occurs, with $\dot{V}_E$ decreased, principally due to a reduction in breath frequency. $\dot{V}O_2$ falls, producing an O_2 debt (although some disagreement exists here), dependent upon work level.
>
> There is a greater effect with expiratory resistance than with inspiratory, principally because exhalation is normally a passive process.

*$\Delta\dot{V}_E$ - increase in expired minute volume
ΔV_d - increase in volume of dead space

The phase of the respiratory cycle with added resistance will have a decreased maximal flow level (flattened) and be prolonged while the other phase will be reduced in time and have higher flow levels.

Ventilation effects are either a function of total added resistance to air flow or the ratio between the inspiratory and expiratory resistance.

Compensation to minimize respiratory effort occurs by a shift in respiratory rate and respiratory level (FRC). There is a tendency for increased PCO_2 in the blood when compensation is incomplete.

Pulmonary compliance will decrease with sufficient added resistance to air flow.

There is a disagreement on whether abnormality of distribution of ventilation occurs.

Subjective responses as indices are not adequate because physiological changes occur at levels lower than can be detected subjectively, although these responses may be well correlated with the introduction of higher levels of resistance to air flow.

There is much disagreement on the air flow resistance levels used as standards which are not to be exceeded, for both inspiration and expiration.

There is disagreement on the flow level at which resistance should be measured (e.g., Drinker, Miura, and Comte believe 120 L/min is a more realistic value than 85 L/min).

Comparability of results of different investigations is difficult because resistance to air flow increases rapidly with exercise in a partially unpredictable, nonlinear relationship.

Although determination of actual added respiratory work is desirable, measurement of resistance at specific flow levels are preferred because of simplicity in evaluation.

The effects of added equipment (external) dead space is summarized as follows:

Although some studies have dealt only with high percentages of inhaled CO_2 and there is some disagreement on the level at which significant effects are obtained, there seems to be evidence showing that the increased inhaled CO_2 does produce undesirable effects at levels expected in respiratory protective devices.

Whenever more than 100 ml dead space is encountered in a mask, an increase in ventilation is expected to maintain a normal alveolar PCO_2, at least under sedentary conditions.

As exercise increases, increased CO_2 concentrations are necessary to stimulate the respiratory center to produce ventilation in excess of that required under normal conditions. When this stimulation does occur, excess ventilation, in an attempt to restore the $PACO_2$ to normal levels, takes place. The tidal volume increases to about 70% of the vital capacity and thereafter breath frequency rises.

This compensatory mechanism seems to be effective when exposed to up to about 2% CO_2, although long exposure will also produce clinical symptoms at lower concentrations.

Some excess ventilation response may also be obtained with as low as 1.1% inspired CO_2 (1% has been recommended as the upper limit in a suit). The physiologic responses to inspired CO_2 adds to the metabolic energy requirement of the worker. Present mask dead spaces seem to be sufficiently large to produce CO_2 response in workers under some conditions.

EXISTING REGULATIONS

Current Federal regulations specifically state that "the wearer shall not experience undue discomfort because of airflow restriction or other physical or chemical changes in the operation of the apparatus"[1] in low temperature environments. Since the definition of the term "undue" was not provided in paragraph 11.3 of Part II, it becomes necessary to examine the specific requirements for each type respirator. The variability found in breathing resistance standards supports the concept that no specific level has been defined at which no significant acute or long-term adverse physiological effects may occur under any working conditions. The influence of manufacturer's capability still seems to be evident in decisions pertaining to acceptable levels.

The following maximum permissible resistance levels are given in the Federal Register, Part II, March 25, 1972:[1]

I. Self-Contained Breathing Apparatus

- A. Open circuit
  1. Inhalation - 32 mm H_2O at 120 L/min
  2. Exhalation
     - a. Demand type - 25 mm H_2O
     - b. Pressure demand type - not to exceed static pressure by more than 51 mm H_2O Static pressure not to exceed 38 mm H_2O

B. Closed circuit
   1. Inhalation - not to exceed difference between exhalation resistance and 100 mm H_2O
   2. Exhalation - 51 mm H_2O

II. Gas Masks

	Inhalation Initial mm H_2O	Inhalation Final mm H_2O	Exhalation mm H_2O
A. Front or back - mounted	60	75	20
B. Front mounted or back mounted with particulate filter	70	85	20
C. Chin style, without particulate filter	40	55	20
D. Chin style with particulate filter	65	80	20
E. Escape, without particulate filter	60	75	20
F. Escape, with particulate filter	70	85	20

III. Supplied Air Respirators

A. Type A (with motor or hand-operated blower)

Max length of hose	Inhalation mm H_2O	Exhalation mm H_2O
75	38	25
150	64	25
250	89	25
300	102	25

B. Type B (mask with blower)
   1. Inhalation - 33 mm H_2O
   2. Exhalation - 25 mm H_2O

C. Type C (standard air line)
   1. Demand type - inhalation 50 mm H_2O
      exhalation 25 mm H_2O
   2. Pressure - demand type
      a. Static 38 mm H_2O
      b. Inhalation - pressure shall not fall below atmospheric at inhalation of <115 L/min
      c. Exhalation - not to exceed static pressure by more than 51 mm H_2O

IV. Dust, Fume, and Mist Respirators

	Inhalation Initial mm H_2O	Inhalation Final mm H_2O	Exhalation mm H_2O
A. Single use	12	15	15
B. Dust, fume, and mist, with single use filter	30	50	20
C. Dust, fume, and mist, with reusable filter	20	40	20
D. Radon daughter	18	25	15
E. Asbestos dust and mist	18	25	15

V. Chemical Cartridge Respirators

	Inhalation Initial mm H_2O	Inhalation Final mm H_2O	Exhalation mm H_2O
A. For gases, vapors, or gases and vapors	40	45	20
B. For gases, vapors or gases and vapors or dusts, fumes and mists	50	70	20
C. For above and mists of paints, lacquers, and enamels	50	70	20

VI. Pesticide Respirators

	Inhalation Initial mm H_2O	Inhalation Final mm H_2O	Exhalation mm H_2O
A. Gas masks	70	85	20
B. Chin style gas mask	65	80	20
C. Powered air purifying	50	70	20
D. Chemical cartridge	50	70	20

RECOMMENDATIONS

Permissible limits seem to be dependent in part on the equipment in use, rather than strictly upon physiological response factors. Even the maximum permitted exhalation resistance normally varies from 15 to 25 mm H_2O at 85 L/min, and to "51 mm H_2O" (above static pressure) in pressure demand systems. This is evident, even though Burgess and Anderson in 1967[67] stated that an expiratory valve designed to offer only 15 mm H_2O resistance at 85 L/min flow was both desirable and feasible. Standards for resistance to air flow and rebreathed carbon dioxide should be dependent principally on the observed physiological responses to the stress imposed, rather than largely upon subjective response studies and the manufacturer's ability to meet requirements, as has been the case in the past. Objective determination of response is needed to separate the subjective effects from those of other mask characteristics and to determine the level of reduction in the functional reserve which acts as a basis for an optimal health status.

There also seems to be no agreement between countries on permissible standards (Luxon, 1973;[68] Revoir, 1971).[69] A comparison of 20 respirators from 10 countries showed exhalation resistance to vary by a factor of 5 (Revoir, 1971).[69] At that time (1971), the British allowed 20 mm H_2O for low toxicity dust and 32 mm H_2O for fine dust respirators. In 1961, up to 50 mm H_2O (85 L/min) at rest, 25 mm H_2O at light to moderate exercise, and 10 mm H_2O at heavy exercise was considered acceptable by the British for inspiratory flow resistance.[70] The Italians and Germans also seem to have had more stringent requirements than the Americans. The need for uniform performance requirements throughout the world was recognized in 1968 when an ACGIH committee came to that conclusion. Recommendations for standardization of testing procedures have been made.[68]

In addition, few references to interactive effects of different respirator design characteristics have been found in the literature. An examination of this phenomenon is needed since added dead space may modify the response to the added resistance to breathing.

Since worker protection is the ultimate goal for the NIOSH effort, all factors influencing wearer use of respiratory protective apparatus should be examined, and necessary changes made to insure maximum use of these devices. Improvements in design to reduce worker "discomfort" and thereby increase frequency and length of time in use are needed. Current intermittent and short-term use by workers has been demonstrated in coal mine studies (NIOSH Contract with Eastern Associated Coal Corporation, 1974)[4,71] and investigations of paint spraying operations (NIOSH Contract with Bendix Corporation, 1973).[5] A worker, in general, will select the respirator which is readily available, most comfortable, and easy to handle, without regard for its protective performance characteristics (Bendix Corporation, 1973).[5] As stated by Harris (1974),[4] "probably most miners found respirators to be uncomfortable as well as a hindrance when working, and consequently, their use was avoided except when discomfort from dust exceeded that from the respirator." Current awareness of hazards has, however, increased use, and educational programs can improve acceptance by the worker (Aucoin, 1973).[3]

Design factors influencing protective performance and modification of the effective resistance to airflow (e.g., leakage)[72,73] are also in need of improvement. Better designs are necessary to reduce leakage, since only about 60% of human faces will properly fit into half mask respirators and only 80% of human faces will adequately fit into full face respirators (Bevis, personal communication).[7] Attempts to improve respirator fit have been made with the aid of anthropometric studies,[74,75] which may stimulate the production of most respirators in at least two sizes. Past and future NIOSH engineering branch investigations may solve some problems, with continued financial support (Gudeman, NIOSH Symposium, 1976).[6]

Significant improvements in overall respirator design which will markedly reduce resistance to breathing and dead space are viewed as a good solution to some of these problems. Although complete comfort while wearing a respirator is not possible, a considerable increase in worker acceptability will result. One new development for protection against dust (The Dust Helmet, 1976)[76] may eliminate inspiratory resistance and dead space while providing full face protection.

Physiological responses to increased air flow resistance and dead space have been determined by several investigators, but additional studies are needed to monitor responses to the stress under the actual conditions found in various respirators and to resolve disagreements on acceptable standards apparent in the literature. These studies should also examine interactive effects of the added dead space and resistance to air flow encountered in respirator masks. Consideration must be given to any acute and long-term effects of using respirators, not only as they influence worker acceptance, but to prevent the "protective" apparatus from causing harm through the additional stress imposed on the worker. As has been shown by Macklem (1971),[77] abnormal stresses, alone, can alter the alveolar structure and elastic properties of the lung, and airway obstruction can lead to abnormal stress. Chronic hyperinflation in experimental animals can lead to loss of elastic recoil of the lung tissue (Buhain, et al. 1970).[78] Increased resistance to breathing sufficient to cause discomfort after a period of use can also cause more exposure to a toxic dust if the worker replaces the filter too soon as a consequence of the discomfort, since penetration of particulate is greatest when the resistance to airflow is least (Revoir and Yurgilas, 1968).[79]

These effects, however, will have been influenced by the previous physical condition of the worker. Peter Macklem (1971)[77] stated that persons with some emphysema and/or obstructive airway disease are not able to tolerate distention of lung tissue to the same degree as normal individuals. Normal persons can tolerate a wide range of resistances to breathing without lasting effects; a considerable margin of safety exists. Studies of persons with abnormal lung function are needed as an aid in the formulation of medical criteria for the required pre-placement examinations prior to use of respiratory protective equipment in industry.

Recognition of this difficulty encountered by persons with lung function abnormalities has apparently led to inclusion of a requirement for medical examination of the worker. The Federal Regulations (1972)[1] state that "persons should not be assigned to tasks requiring use of respirators unless it has been determined that they are physically able to perform the work and use the equipment. The local physician shall determine what health and physical conditions are pertinent. The respirator users medical status should be reviewed periodically (for instance, annually)." Guidelines for the physical examination are, therefore, needed by physicians. As stated by Tabershaw (1974)[80] there are few references in the literature dealing with medical standards for occupations in which a respiratory hazard may exist. In no instance in the regulations are medical criteria given for employment or transfer. At present, only the FVC (Forced Vital Capacity) and the $FEV_{1.0}$ (volume of expired gas removed in the first second of a forced vital capacity maneuver) are used as criteria for respiratory impairment. Even this spirometric test of lung function can only be found in the medical departments of the largest of industrial plants. In addition, there is evidence that the $FEV_{1.0}$ (or FEV_1/FVC) is a poor indicator of early obstructive airway disease.[81,82,83] Measurements from the middle or last portions of the flow volume relationship during a forced vital capacity test seem to be more sensitive indicators,[81,84] although some disagreements still exist about the relative value of these indices. Physiological responses to wearing respirators in both normal persons and in those with emphysema and/or obstructive airway disease must be determined as an aid in formulating medical criteria for pre-placement examinations in industry. There appears to have been no studies of response to respirators using subjects with abnormal lung function. Very little information is available, in general, on physiological limitations (both pulmonary and cardiovascular) to respirator use.[85]

Standards for airflow resistance imposed by the respirator at all applicable levels of work (related to flow level, for simplicity of test procedures) and for permissible equipment dead space, based on both separate and interactive effects of use, is needed to insure that future respirator designs will not impose a significant additional load (stress) on the worker.

This will prevent reductions in working capacity, acute or possible long-term adverse tissue change resulting in impaired body function, and will especially lead to improved acceptance by the worker resulting in increased use and consequent greater protection against exposure to hazardous sustances in the working environment.

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