THE RELATIONSHIP OF OXYGEN COST OF BREATHING TO RESPIRATORY MECHANICAL WORK AND RESPIRATORY FORCE

By MAURICE McGREGOR* AND MARGARET R. BECKLAKE

(From the Joint Cardio-respiratory Service of the Royal Victoria Hospital and the Montreal Children's Hospital, Department of Medicine, McGill University, Montreal, Canada)

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The oxygen cost of increased respiratory activity can be measured by subtracting the oxygen consumption at rest from that observed during the increased respiratory activity. The efficiency of the system can be assessed when the increased respiratory mechanical work is related to the oxygen cost.

Previous estimates of efficiency in man have varied very widely, possibly because very different forms of respiratory work have been employed. Some workers have increased the pressure component of respiratory work by additions to the external air flow resistance, a procedure which will be referred to hereafter as "resistance breathing." In other studies, the volume component of respiratory work has been augmented by increasing the minute volume, without any addition of airway resistance: this will be referred to as "hyperventilation." No investigator employing one experimental technique has studied the oxygen cost of both of these forms of activity.

The first objective of this study was to ascertain whether the relationship of oxygen cost to mechanical respiratory work is the same for hyperventilation as for resistance breathing in normal subjects. The second was to test whether the same relationships hold in the presence of diseases which alter the mechanical properties of the lung.

There is evidence, however, that mechanical work is not the most revealing parameter with which to compare energy consumption. For example, the support of a heavy object at a fixed distance from the ground involves energy consumption but results in no measurable mechanical work, although it is likely that the increased energy consumption would relate to the weight of the object and the length of time it was supported. It was considered of interest to establish the relationship of oxygen consumption of the respiratory muscles to the force exerted by them on the lung.

METHOD

Five healthy volunteer sedentary workers, 5 subjects with obstructive respiratory disease, and 4 cardiac patients were selected for study. Selection of patients was determined by the presence of abnormalities of the mechanical properties of the lungs (Table I). All the patients with obstructive lung disease had moderately severe impairment of exercise capacity except Subject 6 in whom impairment was mild. The two subjects with mitral stenosis had moderate functional disability meriting surgical intervention, while the patients with mitral regurgitation and atrial septal defect suffered from no more than minimal limitation of exercise tolerance. The physical characteristics and results of routine lung function tests are listed in Table I.

Pressure was measured in the esophagus with an esophageal balloon and differential transformer manometer [1] with subjects seated and leaning slightly forward (4). Although usually adjusted to record the difference between esophageal and atmospheric pressure, transpulmonary pressure (differential pressure between esophagus and mouth) also could be observed (see Figure 1). It was assumed that the esophageal balloon recorded the instantaneous average pressure over the outer surface of both lungs.

Respiratory volume was recorded with a sensitive spirometer [2] coupled to a potentiometer, the output of which, together with that of the esophageal pressure gauge, was recorded on a laboratory polygraph. The circuit (Figure 1) consisted of a mouthpiece and low-resistance box valve (5) connected to the oxygen-filled spirometer, so that during control observations all of the expired gas passed through the soda-lime absorber. During hyperventilation an adjustment to the three-way stopcock, T_1, deflected expired gas to the inspiratory line so that expired gas was now partially rebreathed. By selection of a dead space of appropriate size, overbreathing could be achieved without discomfort or faintness resulting from hypocapnea. During resistance breathing the continuity of the circuit was restored, but stopcocks T_1 and T_2

* Research Associate of the National Research Council of Canada.

[1] No. 267B, Sanborn Company, Dover, Mass.

[2] Stead-Wells, Warren E. Collins, Inc., Boston, Mass.

TABLE I

Physical characteristics and respiratory function values *

Subj.	Sex	Diagnosis	Age	Ht	Wt	MMF	Mix.	FRC	VC	Comp.	Resistance In	Resistance Out
			yrs	*cm*	*kg*	*L/sec*	%	*L*	*L*	*L/cm H_2O*	*cm H_2O/L/sec*	
1	M	Normal	39	175	73	3.84	74	4.58	4.22	0.17	1.77	2.29
2	M	Normal	44	173	75	2.80	52	2.91	4.45	0.25	4.20	3.50
3	M	Normal	34	178	73	3.48	77	3.35	5.30	0.28	2.97	4.20
4	M	Normal	30	179	80	4.58	68	3.12	4.64	0.18	2.25	2.72
5	F	Normal	28	160	53	4.70	50	2.45	2.92	0.13	0.91	0.96
6	M	Emphysema	66	166	63	0.21	35	3.74	1.58	0.25	6.30	8.90
7	M	Emphysema	49	159	47	0.15	35	5.92	1.47	0.20	11.80	19.80
8	M	Emphysema	59	173	67	0.30	30	2.56	2.02	0.18	10.00	21.80
9	M	Asthma	50	168	69	0.30	32	6.32	1.92	0.10	15.10	21.30
10	F	Asthma	44	157	66	0.48	57	1.76	1.70	0.08	10.80	17.30
11	F	MS	51	163	42	0.40	56	3.07	1.53	0.11	5.50	9.92
12	F	MS	31	161	67			2.60		0.05	9.18	20.10
13	M	MI	19	178	70	5.70	49	2.83	3.94	0.11	3.80	6.60
14	F	ASD	22	155	35	2.07	77	2.42	2.20	0.06	6.10	12.50

* MS = mitral stenosis;
ASD = inter-atrial septal defect;
MI = mitral incompetence;
MMF = maximal mid-expiratory flow (1);
Mix. = mixing index (2);
FRC = functional residual capacity (2); VC = vital capacity;
Comp. = pulmonary compliance (3).

were adjusted so that inspired and expired gas had to pass through the resistances.

Pressure flow characteristics of the apparatus are illustrated in Figure 2. All tubing was of 32 mm internal bore; thus, resistance to flow was small during control observations and hyperventilation. During resistance breathing the added resistances consisted of lengths (32 cm and 79 cm) of rubber tubing of 5.0 mm internal bore.

Method of procedure was as follows. After a fast of at least 4 hours, the subjects rested in a chair for 30 minutes, breathed pure oxygen for 5 minutes, and then underwent initial control observations for 8 to 12 minutes.

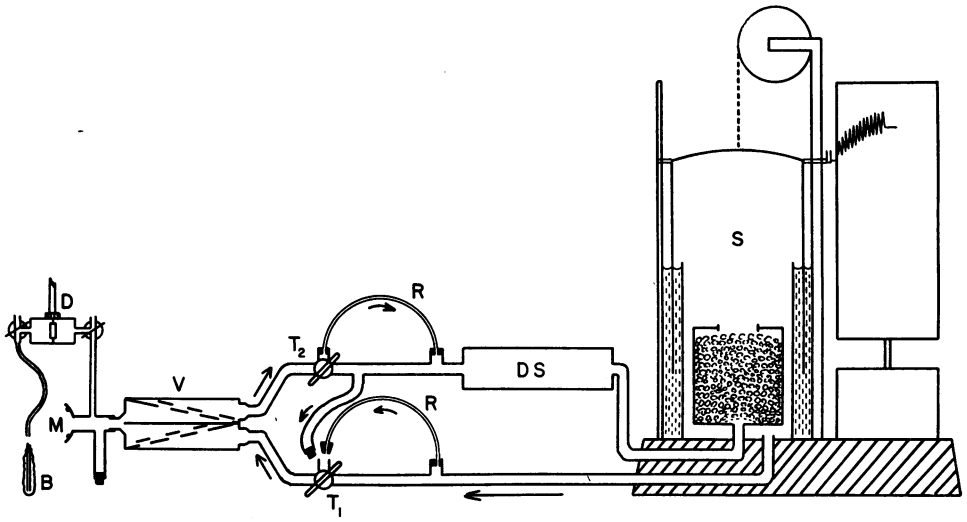

FIG. 1. THE APPARATUS. D = differential manometer; M = mouthpiece and sputum trap; V = low resistance box valve (5); DS = adjustable deadspace; S = Stead-Wells spirometer, with soda-lime absorber; R = variable resistances; B = esophageal balloon; T_1, T_2 = wide-bore three-way stopcocks.

Thereafter, increased respiratory activity was initiated in one of the two ways mentioned. For hyperventilation the subject voluntarily increased his ventilation for 1 to 3 minutes, the apparatus being adjusted for rebreathing. Continuity of the circuit was then restored and control observations again made for a further 8 to 12 minutes. Resistance breathing was likewise preceded and followed by periods of control observation. During the 1 to 3 minutes during which the normal subjects inspired and expired through added resistances, they were instructed to use sufficient effort to maintain the ventilatory volume at normal levels. Unlike the normal subjects it was found that none of the patients could tolerate resistance breathing.

Oxygen consumption during control periods before and after the period of increased ventilatory activity was derived from the slope of the spirometer tracing. The lines describing these slopes, although parallel, do not meet when projected through the period of increased respiratory work but are separated by a distance equivalent to the additional oxygen consumed by the increased work (6, 7). When the oxygen consumption of the second control period differs from the first, the lines describing each are not parallel and the distance between them can no longer be used to determine the increase of oxygen uptake during the period of increased respiratory work. An arbitrary decision was made to exclude all studies in which the control oxygen uptake values before and after the period of increased respiratory activity differed by more than 20 ml per minute. Oxygen uptake values were expressed as STPD, lung volume measurements as APTS, and the breath volume used for computing the respiratory mechanical work was measured at room temperature and pressure.

Mechanical respiratory work per breath was estimated from the area of the loop which resulted from plotting instantaneous esophageal pressure and volume with the addition of that portion of a triangle describing work against lung elastic forces which fell outside this loop. The base of the triangle describing the elastic forces was plotted at the end-expiratory pressure level, not, as is usual, at atmospheric pressure. Because data are based on *differences* between control periods and periods of increased respiratory work, this method of plotting the pressure volume diagram seemed permissible and had the advantage of avoiding the considerable error which may result when *absolute* levels of pleural pressure, as distinct from respiratory fluctuation, are derived from esophageal pressure measurements (3). The product of the average work for 6 to 12 breaths and the frequency gave the respiratory work expressed as kilogram-meters per minute.

The work thus measured, therefore, represents the work performed by the chest cage and diaphragm on the lung and its contents. It includes the work of the abdominal and other accessory muscles during increased work. It does not include the work of the respiratory muscles on the chest cage itself and on the abdominal viscera.

The efficiency of this system was estimated by expressing the increase in mechanical respiratory work, as defined above, as a percentage of the energy equivalent (kilogram-meters per minute) of its increased oxygen consumption.[3]

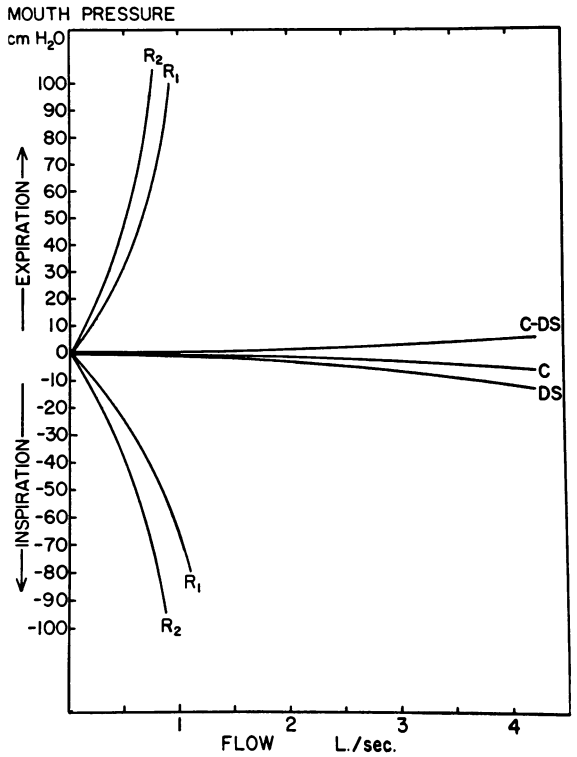

FIG. 2. PRESSURE FLOW RELATIONSHIPS FOR THE INSTRUMENT UNDER CONDITIONS OF USE. C = control observations; DS = hyperventilation through deadspace; R_1, R_2 = resistance breathing. During expiration, C and DS coincide.

Respiratory force was defined as the force exerted, at any instant, by the chest and abdominal muscles on the lung surface, the area of which includes the diaphragmatic and thoracic surfaces of both lungs, but not the mediastinal surface. It was impossible to measure this area but, as a first approximation, it was assumed to equal the outer surface of a solid hemisphere equal in volume to the functional residual capacity. Force per breath was obtained by the product of the mean esophageal pressure per breath (as defined later) and this area. The resultant values depend too greatly upon approximations to be accepted as defining with precision, in kilograms, the actual force exerted. However, inferences based on changes in these values rather than on the values themselves are valid.

Esophageal pressure per breath was measured from the pressure curve. For this purpose it was assumed that the end-expiratory esophageal pressure was the result of the balanced elastic recoil of the chest and lung; change of pressure from this value resulted from the application

[3] A respiratory quotient of 0.8 was assumed.

of force by the respiratory muscles. Change of esophageal pressure from this reference level (irrespective of sign) was thus integrated against time for the whole breath. It will be referred to subsequently as the "mean esophageal pressure" and it should be noted that this term does not refer to an arithmetical mean. Control pressure values and work loops were derived from the average of 6 to 12 breaths in the two periods of resting breathing before and after the period of increased respiratory activity. Values during increased respiratory activity were averaged from 6 to 12 breaths during each of the 1 to 3 minutes of hyperventilation or resistance breathing.

RESULTS

During resting ventilation the average respiratory mechanical work performed by the patients with lung disease was 2.7 times greater than that performed by normal subjects. The average value for the cardiac subjects was similar to that of the normals. Values for respiratory force showed the same trends with greater separation between the groups (Table II).

During hyperventilation the normal and cardiac subjects achieved greater increments of ventilation at a lower oxygen cost per liter ventilated than did the patients with lung disease. Average values for normal and cardiac subjects were 2.23 and 2.26 ml oxygen per liter ventilation, respectively. The equivalent value for patients with lung disease was 21.43 ml per L (Table III). These, with one exception, failed to double their resting ventilatory level in spite of considerable effort. The oxygen cost per kilogram-meter respiratory mechanical work was, on the average, twice as great in the patients with chest disease. Average efficiency as defined for normal, cardiac and respiratory subjects was 3.2, 3.4 and 1.6 per cent, respectively. When related to respiratory force, however, with the exception of Patient 6, the oxygen cost per kilogram was similar in the three groups.

Resistance breathing, which could only be performed by the normal subjects, gave results which were strikingly different from those obtained during hyperventilation (Table IV). The oxygen cost per kilogram-meter respiratory mechanical work was higher during resistance breathing (average efficiency 1 per cent). By contrast, the cost per kilogram respiratory force for this form of activity was lower.

When the data relating oxygen cost to respiratory work for normal and cardiac subjects were plotted (Figure 3A) it was apparent that they were indistinguishable, falling in two distinct slopes for resistance breathing and hyperventila-

TABLE II

Average resting (control) observations *

Subj.	Diagnosis	No. of obs.	f		V_T		$\dot{V}$		R. force		R. mech. work	
			Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
							L/min		*kg*		*kg-m/min*	
1	Normal	12	15.9	0.99	0.58	0.030	9.3	0.99	1.50	0.276	0.22	0.034
2	Normal	12	12.5	0.42	0.95	0.246	11.6	1.92	1.76	0.667	0.54	0.253
3	Normal	6	10.9	1.66	1.03	0.182	10.9	1.07	1.41	0.608	0.44	0.123
4	Normal	6	14.6	0.79	0.77	0.047	11.2	1.01	1.55	0.110	0.35	0.014
5	Normal	6	16.5	1.93	0.47	0.023	7.7	0.73	1.26	0.141	0.75	0.664
	Mean		14.08		0.76		10.14		1.50		0.46	
6	Emph.	2	20.0		0.40		8.0		5.60		0.71	
7	Emph.	2	17.3		0.51		8.8		7.93		1.23	
8	Emph. fib.	2	19.0		0.60		11.4		5.86		1.65	
9	Asthma	4	16.0	0.58	0.68	0.095	12.0	1.09	10.80	0.024	1.66	
10	Asthma	2	13.0		0.48		6.2		4.82		1.16	
	Mean		17.06		0.53		9.28		7.00		1.28	
11	MS	2	14.0		0.53		7.4		3.27		0.51	
12	MS	2	12.0		0.60		7.2		2.94		0.49	
13	MI	2	24.0		0.48		11.5		1.74		0.41	
14	ASD	4	19.0	1.15	0.85	0.046	8.7	0.340	1.71	0.208	1.00	
	Mean		17.25		0.62		8.70		2.41		0.60	

* f = Respiratory frequency; V_T = tidal volume.

TABLE III

Changes in ventilation, oxygen uptake, respiratory mechanical work and respiratory force resulting from unobstructed hyperventilation in all subjects

Subj.	Diagnosis	f	V_T	$\Delta \dot{V}$	Δ R. mech. work	Δ R. force	$\Delta \dot{V}_{O_2}$	Oxygen cost		
									R. mech. work	R. force
				L/min	*kg-m/min*	*kg/min*	*L/min*	*ml O_2/L vent.*	*ml O_2/kg-m*	*ml O_2/kg*
1	Normal	23	1.11	16.4	1.14	2.10	0.022	1.34	19.30	10.48
		21	1.59	22.3	2.77	3.68	0.035	1.57	12.64	9.51
		27	1.66	36.2	2.28	4.97	0.048	1.33	21.05	9.66
2	Normal	13	3.50	34.9	9.30	12.26	0.150	4.30	16.12	12.23
		18	2.83	36.0	21.34	17.47	0.200	5.55	9.37	11.45
		120	0.70	71.6	55.96	31.00	0.313	4.40	5.59	10.10
3	Normal	14	0.52	21.6	1.61	4.34	0.047	2.18	29.19	10.83
		16	1.35	24.2	2.84	5.72	0.055	2.27	19.36	9.62
4	Normal	27	2.31	50.4	10.70	9.59	0.115	2.28	10.75	11.99
5	Normal	23	1.80	34.2	14.79	12.04	0.128	3.74	8.65	10.63
		43	1.40	51.4	13.09	11.70	0.175	3.40	13.37	14.96
	Mean							2.23	15.04	11.05
6	Emphysema	25	0.91	14.9	2.20	2.83	0.072	4.90	32.73	25.44
7	Emphysema	15	0.82	3.5	4.15	24.07	0.230	65.71	55.42	9.55
8	Emphysema	23	0.85	8.1	12.05	31.55	0.202	24.93	16.76	6.40
9	Asthma	40	0.69	16.6	4.56	13.68	0.158	9.52	34.65	11.55
		42	0.57	10.0	6.63	24.58	0.197	19.70	29.71	8.01
10	Asthma	25	1.00	19.0	5.82	5.27	0.073	3.84	12.54	13.85
	Mean							21.43	30.47	12.47
11	MS	30	1.00	22.6	4.87	6.60	0.045	1.99	9.24	6.82
12	MS	28	1.20	26.4	6.76	5.00	0.069	2.61	10.20	13.80
13	MI	32	1.40	33.3	7.64	6.38	0.085	2.55	11.13	13.32
14	ASD	46	1.12	43.0	3.08	6.33	0.081	1.88	26.30	12.80
	Mean							2.26	14.22	11.69

TABLE IV

Changes in ventilation, oxygen uptake, respiratory mechanical work and respiratory force resulting from resistance breathing in normal subjects

Subj.	f	V_T	$\Delta \dot{V}$	Δ R. mech. work	Δ R. force	$\Delta \dot{V}_{O_2}$	Oxygen cost	
							R. mech. work	R. force
			L/min	*kg-m/min*	*kg/min*	*L/min*	*ml O_2/kg-m*	*ml O_2/kg*
1	15	0.71	2.2	2.08	13.10	0.070	33.65	5.34
	17	0.80	4.4	2.43	29.03	0.131	53.90	4.51
	16	0.74	3.7	5.05	25.29	0.153	30.30	6.04
2	6	2.15	1.2	3.26	16.41	0.120	36.81	7.31
	8	1.84	7.5	7.33	28.33	0.216	29.47	7.62
	18	0.55	−4.1	7.89	31.80	0.304	38.53	9.56
3	10	0.80	−2.7	0.18	5.13	0.033	183.30	6.43
	6	1.28	−4.2	1.10	10.97	0.065	59.09	5.93
4	20	0.70	3.7	6.57	26.95	0.139	21.16	5.16
	13	0.91	−0.2	5.21	28.68	0.150	28.79	5.23
5	12	0.66	−0.6	2.86	9.66	0.088	30.77	9.11
Mean							49.62	6.57

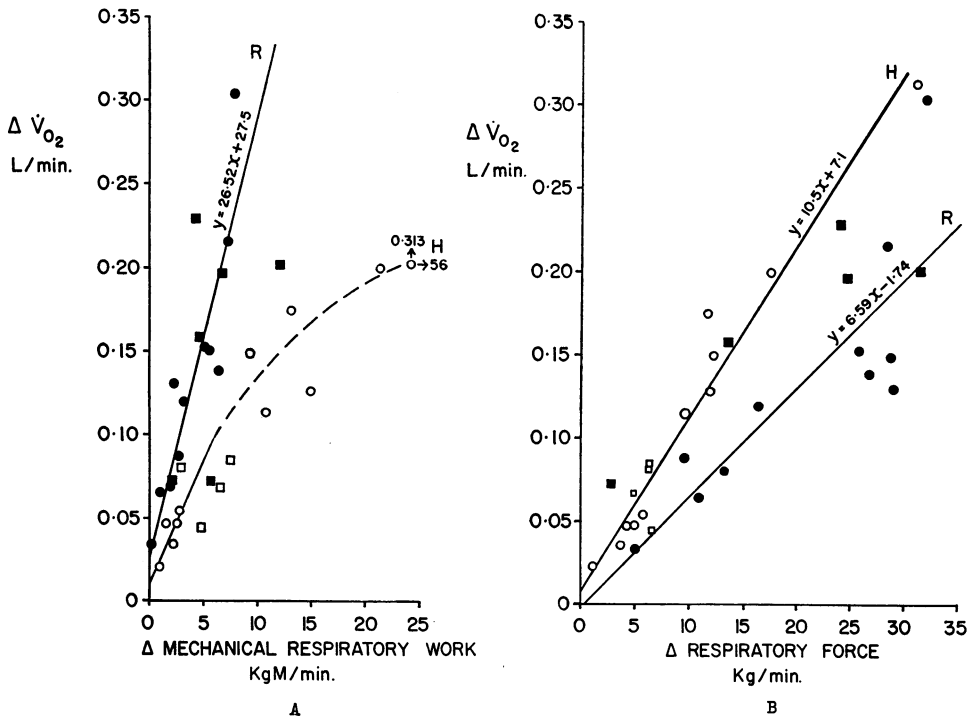

FIG. 3. THE RELATIONSHIP OF OXYGEN COST TO RESPIRATORY MECHANICAL WORK (A) AND TO AN INDEX OF RESPIRATORY FORCE (B). The regression equations are based on the data for normal subjects. ○ = normal subjects during hyperventilation (H); □ = patients with heart disease during hyperventilation; ● = normal subjects during resistance breathing (R); ■ = patients with obstructive lung disease during hyperventilation.

tion, respectively. The patients with obstructive lung disease, however, who in spite of attempted hyperventilation could achieve little increase in ventilatory volume, gave values comparable to those of the normal subjects performing resistance breathing. When oxygen cost was plotted against force (Figure 3B) the normal and cardiac subjects were again comparable, although resistance breathing was achieved at a lower cost than hyperventilation. The data for the patients with lung disease were widely scattered.

DISCUSSION

A possible objection to the validity of these measurements is that major changes in lung volume during the period of increased respiratory activity would tend to alter the area of lung over which pressure was being exerted, and hence produce error in the computation of "force." As judged by shifts in the end-expiratory level of the spirometer tracing, however, these changes were not great. During hyperventilation, normal and cardiac subjects behaved similarly with an average reduction of functional residual capacity of 260 ml (range, 100 to 500 ml). As might be expected, the subjects with obstructive pulmonary disease tended to trap air during hyperventilation with an average increase of functional residual capacity of 200 ml (range, − 100 to + 600 ml). During resistance breathing the end-expiratory level tended to change from breath to breath, the average change in functional residual capacity in normal subjects being an increase of 111 ml (range, − 400 to + 600 ml).

Previous estimations of the efficiency of respiration in normal subjects have shown considerable variation, from as low as 1 per cent (6) to as high as 25 per cent (8). Such discrepancies may be due to differences in the methods of measurement of oxygen cost and of respiratory mechanical work and to differences in the nature of increased respiratory activity.

In most previous studies the oxygen uptake has been measured during prolonged periods (7 to 40 minutes) of increased respiratory activity aimed

at achieving a steady state (8–10). Others, as in the present study, have used short (1 to 3 minutes) bursts of increased respiratory activity (6, 7). The latter method makes possible the study of much higher levels of respiratory work, and there is no reason to believe the results are less accurate. Thus, the oxygen cost observed in this study, when compared with ventilatory volume, is comparable to the values obtained by steady state methods for equivalent degrees of hyperventilation (9–11). Therefore, divergent values for efficiency are unlikely to be due largely to differences in measuring the oxygen uptake—they are more likely due to differences in the method of measurement of respiratory mechanical work.

The efficiency of the combined chest and lung for external resistance work, assessed in three studies (6, 7, 10), gave results of 1 to 10 per cent. In two studies (8, 9), respiratory mechanical work estimates during unobstructed hyperventilation were derived from the area of loops obtained by plotting esophageal pressure against respiratory volume, as in the present study. The values for efficiency for hyperventilating normal subjects obtained in the present study (average, 3.19 per cent) agreed with those in one of these reports (9) but are well below those (19 to 25 per cent) reported in the other (8). A possible reason for this discrepancy is the use of the supine posture by the latter workers. Mead and Gaensler (3) have reported that the esophageal pressure fluctuation may exceed the simultaneous pleural pressure by as much as 60 per cent in the supine position, an artifact which might lead to very considerable overestimation of the mechanical respiratory work.

Irrespective of the absolute values for efficiency obtained in this study, however, it is clear that when the same method is employed for estimating efficiency during both hyperventilation and resistance breathing, the efficiency during the former activity is greater. It should be recalled, however, that in the present study the efficiency of the chest as a whole has been considered. Mechanical work measured did not include that of moving the chest cage or the abdominal contents. For resistance breathing, during which resting ventilatory levels were maintained, there would be no significant increase in the energy expended in moving the chest and abdominal structures so that the calculated efficiency would be unaffected. During hyperventilation, however, unmeasured work was performed on these structures, resulting in an underestimate of the efficiency of the respiratory muscles. If this additional mechanical work could be measured and included in the estimation of efficiency, the discrepancy between resistance breathing and hyperventilation would be even further increased. Thus, in Figure 3A the resistance plots would be unchanged, but those representing hyperventilation would be displaced further to the right.

Consideration of the data for respiratory force, however, is very different. This term likewise described the force applied by the intact chest, diaphragm and abdominal muscles to the lung's surface. Force supplied by the respiratory muscles to the ribs and abdominal structures was not measured. As before, this omission would not affect the relationship of force to oxygen uptake during resistance breathing, but would cause an underestimate of the force exerted by the respiratory muscles during hyperventilation.

If the force exerted by the respiratory muscles on the chest and abdominal structures could be measured and included, the line in Figure 3B describing observations made during hyperventilation would be displaced to the right; that is, nearer to the line describing resistance breathing. In fact, it seemed possible that the discrepancy between the regression equations describing resistance breathing and hyperventilation might be an index of the oxygen cost of moving the chest wall itself.

To check the validity of this hypothesis in Subject 1, measurements were made of the compliance and resistance of the chest wall and lung both separately and together.[4] From the instantaneous volume and flow values, the pressure necessary to deflect the chest wall was computed from instant to instant and added with the appropriate sign to the esophageal pressures recorded in the previous studies. The resulting data for this subject are plotted in Figure 4. In Figure 4A, it can be seen again that the oxygen cost of hyperventilation per

[4] Chest wall compliance = 0.190 ml per cm H_2O; chest wall resistance = 0.6 cm per H_2O per L per second. Measurements were kindly made by Dr. Jere Mead at the Harvard School of Public Health, by a technique reported elsewhere (12).

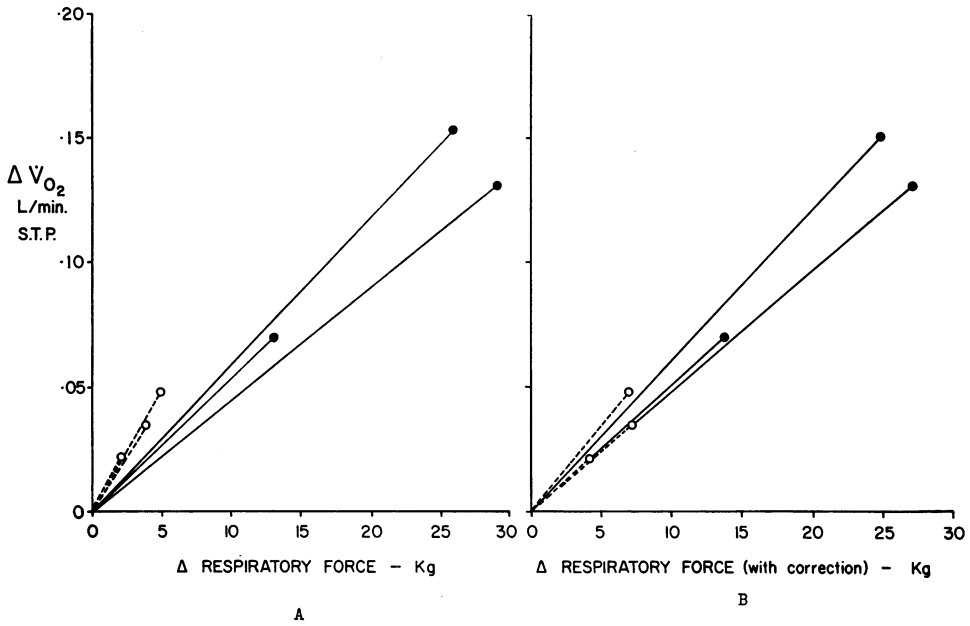

FIG. 4. A) THE RELATIONSHIP OF OXYGEN COST TO RESPIRATORY FORCE EXERTED ON THE LUNG IN SUBJECT 1. B) THE RELATIONSHIP OF OXYGEN COST TO RESPIRATORY FORCE EXERTED ON THE LUNG WITH THE ADDITION OF THE ESTIMATED FORCE EXERTED ON THE CHEST WALL IN SUBJECT 1. ○ = hyperventilation; ● = resistance breathing.

unit force appears greater than that of resistance breathing. With addition of the component of force estimated to be necessary to move the chest wall, the slopes for resistance breathing and hyperventilation cannot be distinguished (Figure 4B).

This suggests that the relationship between oxygen cost and respiratory muscle force may be represented by two slopes which will diverge according to the amount of force which is exerted against the chest and abdominal structures. In other words, the divergence between these slopes (Figure 3B) may represent the additional energy cost of moving the chest and abdominal structures during hyperventilation. No such explanation, however, could account for the differences observed in oxygen cost per unit respiratory mechanical work for the two forms of respiratory activity (Figure 3A).

The observations made on patients with heart and lung disease are too few for any but tentative conclusions. The oxygen cost of hyperventilation in the subjects with heart disease was indistinguishable from that of normal subjects in spite of the fact that the cardiac subjects, although not severely incapacitated, had detectable abnormalities of lung mechanics (Table I). It is likely, however, that heart disease of sufficient severity must result in a significant elevation of respiratory oxygen cost, and Cournand and co-workers did demonstrate this in a comparison of one normal subject with one patient suffering from tight mitral stenosis (13). In the presence of obstructive lung disease, however, the oxygen cost of hyperventilation was very high, due primarily to the high levels of mechanical respiratory work necessary for the small ventilatory increase. It was also due in part to the fact that the oxygen cost per unit mechanical respiratory work was increased above normal. Low respiratory efficiency in emphysematous subjects has been reported previously by Cherniack (10) and is supported by the figures of Fritts, Filler, Fishman and Cournand (9), although they did not consider the values to be "greatly different" from normal. There are two possible reasons for the finding of a reduced mechanical efficiency in these subjects. One is inherent in the method by which mechanical work is computed, i.e., the product of esophageal pressure and volume of gas moved. High levels of obstruction of the airway must result in reduction of the ratio of work measured to energy consumed.

As an extreme example, in maximal respiratory effort against a completely obstructed airway the oxygen consumption will be great, mechanical work measured nil, and efficiency, thus, zero. There is no reason, however, for the force measured to fall off in this way even with complete airway obstruction.

An alternative reason for the low mechanical efficiency measured in the subjects with obstructive lung disease is that their respiratory muscles were acting at a mechanical disadvantage due to hyperinflation. Consideration of the force data provides some support for this (Figure 3B). These subjects, while attempting hyperventilation, failed to increase their minute ventilation greatly in spite of considerable effort (solid squares, Figure 3B), and were thus, in effect, performing "resistance" breathing. The oxygen cost per unit force, however, was apparently higher in most instances than that of normal subjects breathing against increased resistance. These findings indicate an increased oxygen consumption per unit force developed and would be consistent with the working of the respiratory muscles at mechanical disadvantage.

The present findings suggest that as a parameter for comparison with energy consumption, force is a more meaningful expression than mechanical work. A similar conclusion has been reached for heart muscle. Thus, it has been shown that, although heart work augmented by the elevation of arterial pressure consumes more oxygen than does work increased by augmenting venous return, the product of force developed and time appears to relate in a linear fashion to myocardial oxygen consumption over a wide range (14–16) whatever method is used to increase heart work.

Force may also relate more significantly than mechanical work to other respiratory parameters. Marshall, Stone and Christie (17), comparing the onset of severe dyspnea in normal, emphysematous and mitral stenotic subjects, found that, although the level of mechanical respiratory work at which dyspnea became limiting differed greatly in the three groups, exercise was curtailed by dyspnea at a comparable level of "respiratory force" in each group. Furthermore, Mead (12) has recently demonstrated that respiratory depth and frequency vary, not according to the minimal respiratory mechanical work necessary to secure alveolar ventilation, but rather with the minimal respiratory force.

Finally, it is clear that the high oxygen cost of hyperventilation in obstructive lung disease is not only due to increased work of breathing but also to an increased oxygen cost per unit work. This is unlikely to be an artifact in the measurement of respiratory work since the oxygen cost per unit force is also high in these subjects.

SUMMARY

In five normal subjects the oxygen cost of breathing was measured during two forms of increased respiratory activity: unobstructed hyperventilation and breathing in the presence of a high airway resistance. Oxygen cost per unit mechanical respiratory work was greater for resistance breathing than for hyperventilation. Inclusion of the work performed on the chest wall would have increased this anomaly.

By contrast, when related to an index of respiratory force, oxygen cost was higher for hyperventilation. When allowance was made for the force necessary to deflect the chest wall in one subject, the relationship between force and oxygen cost was similar for both forms of respiratory activity. Thus, force appeared to be a more logical parameter to compare with energy consumption than was mechanical work.

In four subjects with disturbances of respiratory mechanics due to heart disease the oxygen cost of hyperventilation was normal whether related to ventilatory volume, respiratory mechanical work or respiratory force.

In five subjects with obstructive lung disease the oxygen cost of hyperventilation was high. This was due both to increased mechanical work per unit volume ventilated and to reduced efficiency per unit mechanical respiratory work. Since oxygen cost per unit force was also high it was likely that the low efficiency was the result of the respiratory muscles' working at a mechanical disadvantage.

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