renal excretion takes place during the recovery phase. The first effect of the cortical hormone upon the animal in suprarenal insufficiency is to produce an increased urinary excretion of potassium, nitrogen, and phosphates, followed by retention of sodium and of chlorides.

The ultimate effect of the hormone is the restoration and maintenance of the proper plasma concentrations of potassium and sodium through regulation of their renal excretion. Hence, it plays a predominant rôle in the stabilization of a proper volume of extracellular fluid.

8074 C

Respiratory Exchange of Oxygen and Carbon Dioxide During Rebreathing from a Rubber Bag.

SIDNEY A. GLADSTONE* AND SIMON DACK. (Introduced by B. S. Oppenheimer.)

From the Medical Service of Dr. B. S. Oppenheimer, The Mount Sinai Hospital, New York City.

During rebreathing from a rubber bag, the nose being compressed with a spring clip so that the lungs and bag form a closed system, O_2 and CO_2 diffuse across the pulmonary epithelium, the direction and rate of diffusion depending on the relative tensions of these gases in the blood and alveolar air. This process has been studied in 18 experiments in 2 normal young men. After the subject had sat in a chair for 10 minutes, the metabolic rate was determined. The subject then expired through a side tube to residual air (an alveolar sample being taken) and then rebreathed from the rubber bag 9 times in 22.2 seconds, being guided by spoken directions from an accurately timed phonograph record. Each inspiration emptied the bag and each expiration was as deep as possible. During the rebreathing alveolar samples were drawn into evacuated tubes at the end of breath No. 3, 6, and 9, dividing each experiment into 3 intervals of 8.0, 7.0, and 7.2 seconds in the order named.

The results of the experiments are presented in tabular form. The contents of the rebreathing bag are noted. The initial volume of the lung-bag system is obtained by adding the gas volume of the bag and the residual lung volume of the subject. In most experi-

*Richard and Ella Hunt Sutro Fellow for Cardiovascular Research.

TABLE I.

Subject		O_2 cons. cc./min.	Barometer	Temperature		Volume	Change in volume		Period	O_2 absorbed			CO_2 discharged			R.Q. I	R.Q. II	R.Q. III
	cc.																	
D	air 2400				1	3994	+24	+22	1	89			111			1.25		
					2	3982	—12	—12	2		52			40			.77	
					3	3954	—28	—29	3			38			11			.29
D	air 2400				1	3991	+21	+20	1	93			113			1.22		
					2	3970	—21	—22	2		65			43			.61	
					3	3941	—29	—28	3			37			9			.24
D	air 2280	296	770	20	1	3974	+ 4	+ 1	1	95			96			1.01		
	O_2 120				2	3963	—11	—10	2		49			39			.80	
					3	3938	—25	—24	3			36			12			.33
D	air 2280	300	768	26	1	3976	+ 6	+11	1	92			103			1.12		
	O_2 120				2	3965	—11	—11	2		53			42			.79	
					3	3937	—28	—29	3			38			9			.24
D	air 2280	269	778	25.5	1	3924	—36	—40	1	70			30			.43		
	CO_2 120				2	3885	—39	—41	2		55			14			.25	
					3	3852	—33	—32	3			50			18			.36
D	air 2280	278	775	24	1	3936	—34	—34	1	62			28			.45		
	CO_2 120				2	3903	—33	—33	2		44			11			.25	
					3	3878	—25	—28	3			45			17			.38
D	air 2280	234	775	24	1	3937	—33	—33	1	56			25			.45		
	CO_2 120				2	3912	—25	—27	2		47			20			.43	
					3	3879	—33	—31	3			16			—17			
D	air 2280	262	767	27	1	3896	—74	—45	1	71			26			.37		
	CO_2 120				2	3863	—33	—32	2		56			24			.43	
					3	3832	—31	—30	3			18			—12			
D	air 2280	246	766	27	1	3926	—44	—43	1	67			24			.36		
	CO_2 120				2	3888	—38	—28	2		48			20			.42	
					3	3861	—27	—25	3			12			—13			
D	air 2150	294	763	20	1	3882	—88	—95	1	59			—36					
	CO_2 250				2	3825	—57	—55	2		45			—10				
					3	3786	—39	—41	3			38			—3			

TABLE I—Continued.

Subject		O_2 cons. cc./min.	Barometer	Temperature		Volume	Change in volume		Period	O_2 absorbed			CO_2 discharged			R.Q. I	R.Q. II	R.Q. III
	cc.																	
D	air 2150	256	763	21	1	3879	—141	—144	1	73			—71					
	CO_2 300				2	3811	—68	—66	2		45			—21				
					3	3764	—47	—48	3			42			—6			
G	air 2400	301	775	23	1	4015	+45	+43	1	61			104			1.71		
					2	3987	—28	—27	2		65			38			.59	
					3	3931	—56	—37	3			82			45			.55
G	air 2400				1	4013	+43	+33	1	74			107			1.45		
					2	4000	—13	—14	2		45			31			.69	
					3	3936	—37	—35	3			60			25			.42
G	air 2400				1	4015	+45	+44	1	58			102			1.76		
					2	4003	—12	—11	2		54			43			.80	
					3	3966	—37	—38	3			64			26			.41
G	air 2280	279	778	19	1	3936	—34	—32	1	43			11			.26		
	CO_2 120				2	3893	—43	—40	2		56			16			.29	
					3	3834	—59	—60	3			80			20			.25
G	air 2280	299	778	185	1	3937	—33	—33	1	47			14			.30		
	CO_2 120				2	3897	—40	—39	2		55			16			.29	
					3	3868	—29	—40	3			60			20			.33
G	air 2150	267	775	21	1	3868	—102	—105	1	53			—52					
	CO_2 250				2	3795	—73	—72	2		50			—22				
					3	3749	—46	—48	3			59			11			.19
G	air 2150	270	775	22	1	3868	—102	—101	1	57			—44					
	CO_2 250				2	3790	—78	—78	2		56			—21				
					3	3734	—56	—57	3			66			9			.14

ments the initial volume was 2400 + 1570 = 3970 cc. The volume changes in the system were calculated from the changes in percentage of nitrogen which is assumed to take a negligible part in the transepithelial gaseous exchange. The volume changes may also be calculated from the difference of O_2 absorbed and CO_2 discharged. The 2 sets of findings listed side by side are in close agreement with a few exceptions probably due to errors in technique or gas analysis. From the total volume of the system and the percentage of each gas, the amount of each gas at the time of sampling may be calculated, and the change in volume of O_2 and CO_2 for each period. From the latter the R. Q. is calculated.

The findings recorded may be understood if one bears in mind first that the alveolar CO_2 is reduced to less than half its normal value when the residual air is diluted by the first inspiration of 2,400 cc. of air from the bag. This lowering of the alveolar CO_2 quickens the diffusion of CO_2 from the blood to the lungs, but as the alveolar CO_2 rises the rate of diffusion is depressed. This fact has been demonstrated by the previous addition of CO_2 to the bag. Furthermore, the demonstration subsequently made that appreciable amounts of blood recirculate within 10 to 12 seconds (see next paper) after rebreathing has begun, indicates that we are dealing with unaltered "mixed venous blood" only during the first period of these experiments, and that the gaseous tensions of the blood reaching the lungs in the second and third periods will be influenced not only by the deep rapid breathing but by the gaseous content of the bag to which the recirculating blood has been previously exposed. Hence the marked change in O_2 absorption observed in successive intervals although the latter differ only slightly in duration.

From comparison of the amounts of oxygen absorbed in the same subject during the first period it may be seen that the addition of O_2 to the bag contents has little effect on the amount of oxygen absorbed, but the latter is depressed if CO_2 is added. Both of these observations are in keeping with the oxyhemoglobin dissociation curves of human blood.[1]

The rate of oxygen absorption during the first period (8 seconds, *i. e.,* before recirculation begins) is about twice the rate observed during the quiet breathing of the metabolism test which preceded the rebreathing experiment. This is due to the increased blood flow through the lungs produced by the deep rapid breathing.

[1] Bohr, C., Hasselbach, K. A., and Krogh, A., *Skand. Arch. f. Physiol.,* 1904, **16**, 402.

As the rebreathing of air continues, the alveolar CO_2 rises, the discharge of CO_2 decreases; the R. Q. exceeds unity in the first period, but quickly falls in the second and third periods. The total volume of the lung-bag system increases during the first period, decreases thereafter. These volume changes explain the difficulties experienced by certain workers in demonstrating the uniform percentage concentration of an inert gas in a lung-bag system,[2, 3] and also the inadequacy of any mixing criteria which are based on percentage concentration of the inert gas without due consideration of the concomitant changes in total volume.

8075 P

The Factor of Recirculation in Acetylene Method for Determination of Cardiac Output.

SIDNEY A. GLADSTONE.* (Introduced by B. S. Oppenheimer.)

From the Medical Service of Dr. B. S. Oppenheimer, The Mount Sinai Hospital, New York City.

Hamilton, Spradlin, and Saam[1] on the basis of animal experiments have concluded that the results of the acetylene method[2] for determining the cardiac output are vitiated by the return of acetylene to the lungs before the procedure is completed. After using the Marshall-Grollman method for one year,[3] the present writer felt the necessity of studying this question in human subjects. If and when recirculation of acetylene-laden blood occurs in appreciable amounts, it will become manifest by a reduction in the rate of diffusion from the lungs into the blood, a reduction in the diffusion constant K of the system, and a rise in the calculated arterio-venous oxygen difference. In 11 rebreathing experiments on 2 subjects, these data were obtained for successive short intervals during each experiment by drawing several alveolar samples, the breathing and sampling being directed by an accurately timed phonograph record.

2 Lundsgaard, C., and Schierbeck, K., *Am. J. Physiol.*, 1923, **64**, 210.

3 Grollman, A., and Marshall, E. K., *Am. J. Physiol.*, 1928, **86**, 110.

* Richard and Ella Hunt Sutro Fellow for Cardiovascular Research.

1 Hamilton, Spradlin, and Saam, *Am. J. Physiol.*, 1932, **100**, 589.

2 Grollman, A., *The Cardiac Output of Man in Health and Disease*, Baltimore, 1932.

3 Gladstone, S. A., *Arch. Int. Med.*, 1935, **55**, 533.