STUDIES IN CONGESTIVE HEART FAILURE

XIV. Orthopnea: Its Relation to Ventilation, Vital Capacity, Oxygen Saturation and Acid-Base Condition of Arterial and Jugular Blood [1]

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INTRODUCTION

A number of hypotheses have been advanced as to the mechanism of the production of orthopnea. In general, these may be divided into three main groups.

1. *Diminished cerebral blood flow:* Krehl (1916) believed that orthopnea, as well as other types of cardiac dyspnea, is always due to an inefficient interchange of gases between the blood and certain cells of the medulla. He pointed out that slowing of the blood stream was one of the means by which such a deficiency could come about. Sir James Mackenzie (1925) claimed that cardiac dyspnea of all types was primarily due to a deficient cardiac output. Neither supported their contentions by experiments. Recently, Ernstene and Blumgart (1930) investigated the subject, and interpreted their studies as indicating that orthopnea was due to diminished cerebral blood flow. They stated:

"This theory is based on the fact that increased cerebral venous pressure diminishes intracranial blood flow, thereby favoring increased anoxemia of the respiratory center. . . . In the upright position the pressure in the veins about the respiratory center is kept more nearly normal than in any other position and the blood flow in the capillaries feeding these veins is increased. . . . In general it was found that the higher the venous pressure the greater was the orthopnea. When orthopneic patients were placed in the recumbent position with the head flat, simple elevation of the head by flexion of it on the thorax produced, almost without exception, conspicuous diminution of the respiratory distress, but had no significant effect on the

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vital capacity of the lungs. . . . The extent of the diminution of the vital capacity of the lungs does not seem to be an important factor in determining the degree of elevation which gives the patient maximum relief. Orthopnea of necessity was observed in patients in whom the vital capacity of the lungs was not significantly increased by changing from the recumbent to the sitting position."

2. *Deficient aeration of the blood in the lungs.* Haldane, Meakins, and Priestley (1919) pointed out that the expansion of the lungs is uneven in the recumbent posture. In normal subjects this is compensated for by slower and deeper breathing, but in patients with cardiac failure with diminished vital capacity, the depth of breathing cannot be materially increased. Consequently, they believed that the arterial saturation with oxygen was less in the reclining position in such patients and that orthopnea was due to this fact.

Meakins and Davies (1925) believed that decreased arterial saturation in the horizontal position was an important factor in the production of orthopnea. Wilson (1928) confirmed Bohr's (1907) observation that the reserve air was greater in the sitting than in the recumbent posture. Consequently, he believed that orthopnea could be explained by an assumption of decreased arterial saturation. However, none of these authors performed analyses of arterial blood.

3. *Mechanical changes in the lungs.* Bohr, in 1907, showed that the vital capacity and the reserve air were greater in the sitting than in the recumbent position. Rubow (1909) believed that kinking of the veins in the recumbent posture caused increased resistance in the pulmonary circulation. Christie and Beams (1922) found reduction of vital capacity in the recumbent position. The average degree of diminution was 5.5 per cent in two hundred and seventy normal subjects and 26.5 per cent in a series of seven patients with orthopnea. They conclude that the decrease in vital capacity was the cause of the distress felt in the horizontal position. Ernstene and Blumgart (1930) found much smaller difference in vital capacity in their orthopneic patients who had on the average only eight per cent greater vital capacity in the sitting than the recumbent position.

Field and Bock (1925) found diminished cardiac output in the sitting as compared to the recumbent position, and believed that the respiratory distress which comes on lying down is due to pulmonary congestion and consequent diminution in vital capacity. The most recent observations on the subject, those of Grollman (1928), did not demonstrate much change in cardiac output with change of posture.

Blackhall-Morison (1928) believed that the orthopneic position produced benefit by limiting the venous inflow into the heart. Hirschfelder (1913) thought that there were several different factors responsible for the relief experienced in the sitting posture. These were (1) descent of the liver and diaphragm with consequent increase in the air space of the chest; (2) equali-

zation of the load on the two ventricles, because of diminished venous return to the right auricle; (3) diminished venous stasis in the medulla.

Present study

The investigation of the relation of the *dyspnea of exercise* reported in the preceding paper (Cullen, Harrison, Calhoun, Wilkins and Tims (1931)) in which it was found that the changes in the oxygen saturation and acid-base condition of the blood were secondary to and not causative of the dyspnea indicates that similar relations might be true in *orthopnea.*

The concept that orthopnea in patients with cardiac failure is essentially due to decrease in cerebral blood flow seems extremely unlikely to us because it is contrary to the following well known clinical facts:

(1) Syncope, which is presumably due to decreased cerebral blood flow, is often relieved by putting the head down.

(2) Orthopnea occurs in a variety of conditions such as congestive heart failure, massive ascites, pleural effusion, pneumothorax and some cases of pneumonia. All of these conditions are associated with decrease in vital capacity, but only one of them, namely, cardiac failure is regularly associated with increase in venous pressure. Is orthopnea in cardiac disease to be regarded as being entirely different from orthopnea in other conditions?

(3) Orthopnea is invariably absent in hemorrhage and shock which are known to be associated with diminution of the cardiac output.

(4) Orthopnea often occurs in patients with cardiac disease, who have no edema, no enlargement of the liver, and no striking distension of the cervical veins.

In trying to evaluate the experimental data from which the various opinions reviewed here have been deduced, it became evident that more adequate observations were needed in which both the respiratory factors and blood chemical changes were studied in the same patients at the same time. The present paper reports the results of a study planned to furnish such data.

Blood studies. Blood was obtained from the brachial artery and from the internal jugular vein, according to the technique described by Myerson, Halloran and Hirsch (1927) and by Lennox (1930). Analyses for oxygen and carbon dioxide were made, the hydrogen ion

concentration was determined and the carbon dioxide tension was calculated according to the methods described in the preceding paper of this series. (Cullen, Harrison, Calhoun, Wilkins and Tims (1931).)

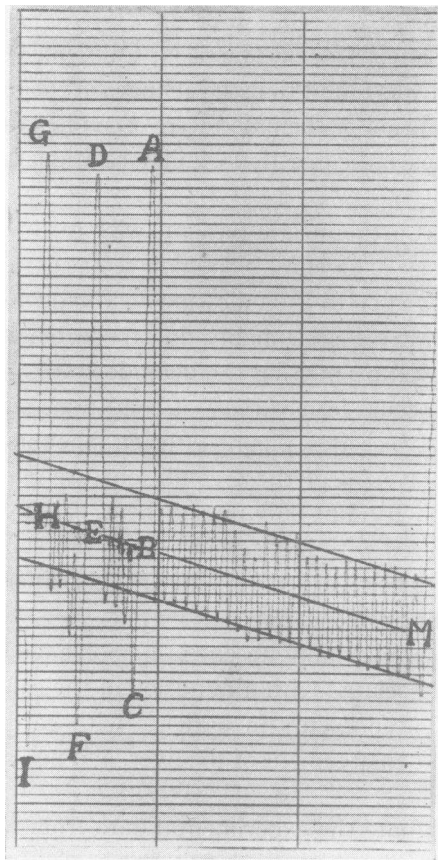

FIG. 1

The curve runs from right to left. The upstroke denotes inspiration; the down stroke expiration. The line M-B-E-H represents the mid-position of the lungs. Three vital capacities were taken: A-B-C, D-E-F and G-H-I. The values for these as calculated from the curve were 249 liters, 260 liters and 281 liters respectively. However, when the greatest of the three inspirations (A-B) is added to the greatest of the three expirations (H-I) the value obtained is 290 liters. It is obvious that this represents the true vital capacity and that the other values are all lower than the true, i.e., maximum vital capacity.

The observations were made on a group of eight patients suffering from different types and degrees of cardiac disease and congestive heart failure. The group includes one patient, J. L., with tuberculosis and asthma as representative of pulmonary complication. The findings in his case are summarized separately from those in the rest of the group. Three normal males were used as controls.

Respiratory measurements were made in the usual way. In measuring ventilation the patient breathed into a Tissot spirometer through a face mask equipped with appropriate valves. The vital capacity for some of the observations were obtained in the usual way but for the later determinations a refinement was introduced.

The subject was connected to a Benedict basal metabolism spirometer and allowed to breathe quietly for a few minutes, long enough to establish the slope of a mid-position line. The subject then made a series of maximal inhalations and exhalations. The deepest inhalation, measured from the mid position was added to the greatest expiration to establish the true maximum vital capacity. (See Fig. 1.)

RESULTS

A. The relation of orthopnea to the oxygen content and acid base balance of the blood

The data are shown in Table 1. The findings in the case of J. L. are discussed separately at the end of this section. The *arterial oxygen saturation* was usually somewhat greater in the sitting than the recumbent position but the change was often slight and sometimes absent. Most of the values in either position were within normal limits. Consequently, it seems extremely unlikely that the benefit derived from the sitting position can be explained by the changes in arterial saturation, although in some cases this may be one factor.

The jugular venous oxygen content was less once, greater once, and practically unchanged in the remaining four instances, in the sitting position as compared to the recumbent posture. *The arteriovenous oxygen difference of the blood passing through the brain* was determined in five instances and was in every case almost exactly the same in the two positions. However, the actual values varied widely in different persons, the lowest being 3.62 and the highest 10.54 volumes per cent O_2. The mean arteriovenous oxygen difference for the blood obtained from

TABLE 1

The relation between acid-base condition of the blood and the ventilation in orthopnea

Subject	Chief diagnosis	Date	Degree of orthopnea	Position	Oxygen capacity	Arterial oxygen content	Arterial oxygen saturation	Jugular venous oxygen content	Arterio-venous oxygen difference	Carbon dioxide content of serum		pH of serum		Carbon dioxide tension of serum		Vital capacity per square meter	Ventilation per minute per square meter	Ventilation per minute / Vital capacity ratio
										Arterial	Jugular	Arterial	Jugular	Arterial	Jugular			
					volumes per cent	*volumes per cent*	*per cent*	*volumes per cent*	*volumes per cent*	*volumes per cent*	*volumes per cent*			*mm. Hg*	*mm. Hg*	*liters*	*liters*	
Al. C.	Syphilitic aortic insufficiency	December 3, 1930	Necessity	Recumbent	13.32			7.99			66.7		7.40		31.7	1.21	6.88	5.68
				Sitting				7.02			67.4		7.46		28.2	1.51	7.14	4.73
		December 12, 1930	Choice	Recumbent	19.12	17.44	91.3			61.5		7.53		32.8		1.56	5.68	3.64
				Sitting		18.75	98.1			58.7		7.52		32.0		2.08	6.12	2.94
P. H.	Hypertension Bronchitis Emphysema	December 16, 1930	Necessity	Recumbent	16.56	15.12	91.3			61.4		7.65		25.0		1.06	4.98	4.70
				Sitting		15.72	95.0			61.6		7.66		24.6		1.30	5.70	4.38
J. P.	Arteriosclerosis	December 9, 1930	Necessity	Recumbent	17.66	16.58	94.0			65.6		7.58		31.3		0.65	4.71	7.24
				Sitting		17.40	98.6			65.5		7.58		31.3		1.04	5.50	5.28
E. G.	Syphilitic aortic insufficiency	December 18, 1930	Choice	Recumbent	14.76	13.56	92.0	9.94	3.62	63.5	68.3	7.49	7.43	37.1	45.4	1.26	5.44	4.32
				Sitting		13.80	93.5	9.94	3.86	62.9	66.4	7.48	7.46	37.5	41.4	1.69	5.32	3.15
J. L.	Tuberculosis Emphysema Asthma	January 7, 1931	Necessity	Recumbent	18.40	12.00	65.2	8.37	3.63	73.9	78.6	7.37	7.28	56.2	72.7	0.75	4.26	5.68
				Sitting		12.36	67.1	8.61	3.75	72.9	78.0	7.46	7.34	45.5	63.2	1.06	3.60	3.40
D. W.	Hypertension	January 16, 1931	Necessity	Recumbent	14.90	13.20	88.6	5.82	7.38	47.8	55.8	7.54	7.46	25.0	34.8	0.68	6.18	9.10
				Sitting		14.40	96.7	6.91	7.49	47.8	56.3	7.55	7.46	24.4	35.1	0.75	6.60	8.80
C. M.	Hypertension	January 19, 1931	Necessity	Recumbent	16.24	14.80	91.1	6.92	7.88	71.8	78.0	7.55	7.47	36.6	47.6	0.78	5.56	6.53
				Sitting		14.55	89.6	6.92	7.63	70.4	79.3	7.56	7.48	35.2	47.3	1.19	5.12	4.67
W. J.	Hypertension Arteriosclerosis	January 23, 1931	Necessity	Recumbent	18.65	17.32	93.0	6.79	10.53	46.8	58.4	7.61	7.59	20.9	27.3	0.92	9.82	10.68
				Sitting		17.45	93.6	6.91	10.54	45.8	58.4	7.67	7.59	17.9	27.3	1.23	7.32	5.95

the internal jugular vein was 6.51 volumes per cent in the recumbent position and 6.55 volumes per cent in the sitting posture. Therefore, it is concluded that orthopnea cannot be due to diminished cerebral blood flow in the recumbent position. The only possible alternatives to this conclusion are the unlikely assumptions either that the portion of the brain drained by the jugular veins is different in the two positions or that the oxygen consumption of the brain is less when an individual lies down than when he sits up.

It may be noted that the average value for the utilization of oxygen in the blood passing through the brain of the patients with cardiac failure was almost exactly the same as those found by Lennox (1930) in his series of fifty-one individuals with no cardiac or pulmonary disease. His average value was 6.5 volumes per cent, but different normal subjects showed marked differences, as did our patients. The idea that cardiac dyspnea is primarily due to diminished cerebral blood flow has been widely accepted. These data, like those published in our preceding paper (Cullen, Harrison, Calhoun, Wilkins and Tims (1931)) fail to support this assumption. Some of our patients were severely decompensated but in only one of the five subjects (W. J.) were the values for jugular oxygen much lower than the normal average. It seems clear that the cause of cardiac dyspnea must be sought elsewhere.

The carbon dioxide content of the serum was often slightly less in the arterial blood in the sitting than in the recumbent position. The difference was not striking, being usually less than one volume per cent, and in three of the eight instances no change was noted. No constant difference in the carbon dioxide content of the jugular blood was observed. All values except one (W. J.) were within normal limits.

The hydrogen ion concentration of the arterial serum was the same—within the limit of error of the method in the two positions, in six of the eight observations. In one instance (W. J.) decided shifts toward alkalinity were observed when the patient sat up. The pH of the blood from the internal jugular vein was the same in the two positions in three observations and more alkaline in the sitting position in three. The arterial pH was above 7.50 (both sitting and recumbent) in six of eight observations and in two of these it was above 7.60. The jugular blood was within normal range with one exception, W. J., pH 7.59.

These findings are in accord with those of other investigators (Eppinger, Kisch and Schwarz (1927); Fraser, Harris, Hilton and Linder (1928)) that the majority of patients with cardiac insufficiency have blood which is toward or beyond the alkaline side of the normal range.

The *carbon dioxide tension* of the arterial blood was practically the same in the two positions in six of eight determinations. In the remaining two subjects the carbon dioxide tension was lower sitting than recumbent. The carbon dioxide tension of the blood from the internal jugular vein was practically the same in the two positions in three patients, was somewhat less sitting in two individuals and was very much less sitting in one patient. The carbon dioxide tension in both positions was rather lower than normal in six of eight patients, and was normal twice.

It is evident from these data that the majority of patients with congestive failure have a slight or moderate alkalemia (tendency toward decreasing hydrogen ion concentration) from over ventilation, although the blood may be normal in regard to its alkali reserve. In none of the patients were the changes in the blood adequate to account entirely for the discomfort in the recumbent position. Their distress could be explained neither by deficient circulation nor by inadequate aeration, and such changes as were found in the blood were evidently effects rather than causes of their dyspnea.

The findings in J. L. are of special interest. This man had tuberculosis; emphysema, asthma and cardiac insufficiency. His ventilation was less than that of any other patient studied, and yet he had more subjective dyspnea than any other subject in the series. (It has been shown previously by Harrison, Turley, Jones and Calhoun (1931) that when respiratory obstruction is present the amount of ventilation is no index to the degree of subjective dyspnea.) He was the only patient of the series with marked anoxemia. The findings in the blood were those of uncompensated carbon dioxide excess and his distress was due to deficient aeration which was reflected in the blood. His carbon dioxide tension was extremely high and much higher lying than sitting. It is evident that, in this type of patient, orthopnea could be explained by the less efficient gas exchange in the lung in the recumbent posture.

B. Respiratory measurements in patients with orthopnea

Vital capacity and ventilation ratio. In order to compare quantitatively the respiratory embarrassment of the patients the values for the ratio $\frac{\text{ventilation per minute}}{\text{vital capacity}}$ have been determined and are shown in Table 2. It has been shown previously (Harrison, Turley, Jones and Calhoun (1931)) that this quotient is a fairly accurate expression of the degree of respiratory distress. In the normal subjects the values are the same in the two positions and range between 1.50 and 1.80, whereas, the values for the patients are much higher, and in the majority of them the figures are considerably less in the sitting than in the recumbent posture. In an attempt to analyze the factors responsible for the greater dyspnea in the recumbent position the respiratory measurements are presented in detail in Table 2.

Observations were made of the ventilation per minute, respiratory rate and vital capacity of nine patients and of three normal subjects in the recumbent and sitting positions. The other data were calculated from these measurements. The total dead space (instrumental plus anatomical) was assumed to be 160 cubic centimeters. The mask used fitted the face closely and did not have a dead space of more than 30 cubic centimeters. The anatomical dead space was assumed to be 130 cc. for each subject. The values for dead space ventilation and alveolar ventilation are therefore to be regarded as only crude approximations.

The order in which the various observations were made was varied, some of the subjects being studied first in the recumbent and then in the sitting position while in other individuals the observations while sitting were made first. For the sake of uniformity in tabulation the measurements in the recumbent posture are in each subject presented first.

The vital capacities of the patients were, as would be expected, much less than those of the normal individuals. The latter had slightly greater vital capacity in the sitting position, the difference being about the same as that found by Christie and Beams (1922) in their large series of normal subjects. In all of the patients the vital capacity was greater in the sitting position and the degree of difference was not only relatively but in most cases was actually greater than in the normal

TABLE 2

Orthopnea in relation to respiratory measurements

Subject	Diagnosis	Surface area	Date	Degree of orthopnea	Position	Vital capacity	Actual change in vital capacity	Change in vital capacity	Respiratory rate	Ventilation per minute	Mean tidal air	Vital capacity used per breath	Dead space ventilation per minute	Alveolar ventilation per minute per square meter	Ventilation per minute / Vital capacity ratio
		square meters				*liters*	*cc.*	*per cent*	*per minute*	*liters*	*cc.*	*per cent*	*liters*	*liters*	
W. E. W.	Normal	1.75	December 10, 1930	None	Recumbent	3.80	250	6.6	13	6.74	519	13.6	2.08	2.66	1.77
					Sitting	4.05			15	7.06	471	11.6	2.40	2.66	1.74
T. R. H.	Normal	1.75	December 10, 1930	None	Recumbent	4.10	350	8.5	8	6.32	790	19.2	1.28	2.88	1.52
					Sitting	4.45			10	6.66	666	15.0	1.60	2.89	1.50
J. A. C.	Normal	1.75	December 10, 1930	None	Recumbent	4.00	350	8.8	10	6.10	610	15.3	1.60	2.57	1.52
					Sitting	4.35			14	6.66	475	10.9	2.24	2.52	1.53
Al. C.	Syphilitic aortic insufficiency	1.66	December 12, 1930	Necessity	Recumbent	2.60	850	32.7	18	9.42	518	19.9	2.88	3.94	3.62
					Sitting	3.45			17	10.16	598	17.3	2.72	4.48	2.94
			January 2, 1931	Choice	Recumbent	2.90	200	6.9	20	9.18	459	15.8	3.20	3.60	3.16
					Sitting	3.10			19	9.14	471	15.2	3.04	3.67	2.95
P. H.	Bronchitis Emphysema Hypertension	1.65	December 11, 1930	Necessity Severe	Recumbent	1.50	550	36.7	32	13.48	421	28.0	5.12	5.06	9.00
					Sitting	2.05			26	11.08	426	20.8	4.16	4.20	5.41
			December 16, 1930	Necessity	Recumbent	1.78	370	20.8	28	8.24	294	16.5	4.48	2.28	4.63
					Sitting	2.15			26	9.40	362	16.8	4.16	3.18	4.37

TABLE 2 (*continued*)

Subject	Diagnosis	Surface area	Date	Degree of orthopnea	Position	Vital capacity	Actual change in vital capacity	Change in vital capacity	Respiratory rate	Ventilation per minute	Mean tidal air	Vital capacity used per breath	Dead space ventilation per minute	Alveolar ventilation per minute per square meter	Ventilation per minute / Vital capacity ratio
		square meter				*liters*	*cc.*	*per cent*	*per minute*	*liters*	*cc.*	*per cent*	*liters*	*liters*	
J. L.	Tuberculosis Asthma Emphysema	1.54	January 7, 1931	Necessity	Recumbent	1.15	480	41.7	19	6.56	345	30.0	3.04	2.28	4.38
					Sitting	1.63			14	5.52	395	24.2	2.24	2.13	3.40
D. W.	Hypertension	1.68	January 16, 1931	Necessity	Recumbent	1.15	100	8.7	22	10.36	471	41.0	3.52	4.07	9.01
					Sitting	1.25			23	11.10	482	38.5	3.68	4.42	8.88
E. G.	Syphilitic	1.69	December 18, 1930	Choice	Recumbent	2.12	730	34.4	29	9.18	312	14.7	4.64	2.69	4.33
					Sitting	2.85			26	9.00	346	12.1	4.16	2.86	3.16
W. M. J.	Hypertension	1.98	January 17, 1931	Necessity	Recumbent	1.90	300	15.8	14	9.44	657	34.6	2.24	3.64	4.96
					Sitting	2.20			14	8.62	616	28.0	2.24	3.22	3.92
A. B.	Hypertension	1.67	January 15, 1931	Choice	Recumbent	1.65	420	25.5	23	8.76	380	23.0	3.68	3.04	5.31
					Sitting	2.07			21	8.32	396	19.1	3.36	2.97	4.02
W. J.	Hypertension Arteriosclerosis	1.63	January 23, 1931	Necessity Severe	Recumbent	1.50	350	23.3	32	16.00	500	33.3	5.12	6.71	10.68
					Sitting	1.85			21	11.92	568	30.5	3.36	5.25	6.44
C. M.	Hypertension Emphysema	1.68	January 19, 1931	Necessity	Recumbent	1.30	700	53.9	23	8.56	372	28.6	3.68	2.90	6.59
					Sitting	2.00			22	9.36	425	21.2	3.52	3.43	4.68

subjects. For the latter the average increase in vital capacity on changing from the recumbent to the sitting posture was 320 cubic centimeters, or eight per cent of the recumbent value. For the patients the average figures were 460 cubic centimeters, or 27 per cent of the recumbent value. Again our data agree with those of Christie and Beams who found an average difference of 26.5 per cent in the two positions in patients with orthopnea. In general the degree of change of vital capacity with change of position paralleled the severity of cardiac failure. In a previous paper of this series (Harrison, Turley, Jones, and Calhoun (1931)) it was shown that when there is no obstruction to breathing, subjective respiratory distress is closely related to the fraction of the vital capacity used in breathing. Regardless of the actual vital capacity a maximal breath requires about the same degree of muscular effort. It can be seen from Table 2 that the patients with cardiac disease usually used a considerably greater fraction of their vital capacity at each breath than did the normal subjects, and this must have been attended with correspondingly greater muscular effort. It seems obvious that decrease in vital capacity, if of sufficient degree, can *per se* cause respiratory distress even if, as shown in this paper, it is not associated with changes in the acid-base balance of the blood. Our patients were, for the most part, somewhat short of breath on sitting, and on lying down the further decrease in vital capacity was apparently a potent factor in causing an increase in their dyspnea.

These conclusions are in agreement with those of Peabody (1916–17) and his co-workers who emphasized the importance of decreased vital capacity in producing orthopnea and those of Field and Bock (1925) who concluded that increased congestion of the lungs in the recumbent position is a cause of orthopnea.

The respiratory rate was usually considerably greater than normal in the patients in both positions. Normal individuals sometimes breathe somewhat faster when sitting than when recumbent (Tables 2 and 3). Haldane's normal men exhibited the same phenomenon, the cause of which is not clear. The patients with cardiac failure did not breathe more rapidly when sitting. In five of fifteen observations their rates were ten per cent or more faster when recumbent. It has been generally assumed that the more rapid breathing in patients with cardiac disease is due to anoxemia or some other change in the composition of

the blood. However, it has already been shown (Table 1) that this is not true of the blood of our patients. For the present it is necessary to leave the problem as to the cause of the rapid breathing unexplained. It seems likely that abnormally rapid respiratory rate in itself tends to increase subjective respiratory distress and thus, regardless of initial cause, set up a vicious cycle.

The minute ventilation was usually greater in the patients than in the normal subjects. In general, the degree of increase in ventilation paralleled the degree of orthopnea. The patient, J. L., with pulmonary disease in addition to cardiac failure was an exception in this regard. He could not increase his ventilation because of respiratory obstruction. The normal subjects had slightly greater ventilation in the sitting posture. The two patients with the severest orthopnea had greater ventilation in the recumbent position. The other patients were inconstant in this regard. The greater minute ventilations in both postures of the subjects with cardiac disease were largely dependent on increased respiratory rates. The alveolar ventilation was high in those two patients whose dyspnea was very severe, but were often approximately normal in other subjects. The dead space ventilation was greater when sitting in the normal subjects, but this was not true in the patients. The alveolar ventilation of the normal subjects was not affected by change of posture, whereas, some of the patients had greater alveolar ventilation in one position and some in the other.

The depth of breathing (tidal air) was usually less than in the normal subjects although this difference was inconstant. The normal subjects breathed deeper when recumbent; the reverse tended to be true of the patients.

It is obvious that such changes as were found in the total ventilation and in the deep space ventilation were in large measure dependent on the increase in respiratory rate.

It is evident that in the sitting position the increase in vital capacity is the most constant factor causing a diminution of the ratio mentioned above and hence of the respiratory distress, but it is also evident that in certain cases the decrease in respiratory rate and consequent decline of dead space ventilation and total ventilation are extremely important factors in the production of the relief experienced in the sitting position.

The effect of posture on the mid-position of the lungs and on the complementary air. It has been emphasized above that patients with

orthopnea may breathe more rapidly in the recumbent than in the sitting posture while normal individuals may breathe slightly slower in the recumbent posture. In both positions the respiratory rate of the patients is usually faster than normal. It has also been shown that the tachypnea of patients with cardiac failure is not due to anoxemia or changes in the acid-base balance of the blood.

That the rate of breathing is closely related to reflexes from the lungs through the vagus nerves was demonstrated more than sixty years ago by Hering and Brauer (1868), who showed that distension of the lungs produced an expiratory movement, whereas, deflation of the lungs produced an inspiratory movement, these effects being absent when the vagus nerves were cut. These observations have been confirmed by numerous observers. It seems possible that if for any reason such as rigidity or congestion, air cannot enter certain parts of the lungs the remaining functioning portion might be distended sufficiently to excite the Hering Brauer reflex with much less air than is normally required and thus bring about an increased rate of breathing. Further data on the nervous control of respiration in relation to changes in the lungs are needed. In the succeeding paper of this series (Harrison, Calhoun, Cullen, Wilkins and Pilcher (1932)) such data will be presented.

Further, the amount of air required to distend a given functioning portion of a lung is more nearly a function of the "complementary air" (volume from mid-position of lung to maximum inspiration)—than of total vital capacity. If the rigidity or congestion which brings about reduced vital capacity is of such a nature that when a subject sits up the mid-position of the lung does not change proportionately as much as in the normal and there results an increased complementary air, one would expect that subject to breathe more slowly. In order to test this reasoning further observations were made. The recumbent subjects were connected to a Benedict spirometer. Vital capacity, complementary air and supplementary air were measured. The subject then breathed normally for three minutes during which the respirations were recorded. Then, without removing the mouthpiece, the subject sat up and the observations were repeated. After a few minutes rest the observations were again made, in this instance the measurements being taken first with the patient sitting. The data are shown in Table 3. The vital capacity in the sitting posture was slightly greater

TABLE 3

The effect of posture on the mid-position of the lungs, the complementary air, and the supplementary air *

Subject and sex	Diagnosis	Position	Respiratory rate	Complementary air	Supplementary air	Vital capacity	Shift in mid-position	Degree of orthopnea
			per minute	*cc.*	*cc.*	*cc.*	*cc.*	
F. J. F.	Normal	Recumbent Sitting	14 14	2010 1720	730 1200	2740 2920	390	None
M. B. F.	Normal	Recumbent Sitting	18 19	1820 1740	750 970	2570 2710	250	None
S. S. F.	Normal	Recumbent Sitting	15 15	2530 2320	1270 1540	3800 3860	240	None
W. E. W. M.	Normal	Recumbent Sitting	15 15	2490 2280	1620 1990	4110 4270	250	None
T. R. H. M.	Normal	Recumbent Sitting	7 8	2840 2630	1400 1630	4230 4260	180	None
W. J. M.	Hypertension Arteriosclerosis Emphysema	Recumbent Sitting	24 25	1350 1140	790 1040	2140 2180	150	None
W. C. M.	Hypertension Arteriosclerosis	Recumbent Sitting	18 17	1850 1850	460 700	2310 2500	150	Choice
L. H. M.	Bronchiectasis Asthma	Recumbent Sitting	14 13	2030 2120	410 580	2440 2700	220	Choice
A. C. M.	Syphilitic aortic insufficiency	Recumbent Sitting	23 23	910 1080	560 910	1470 1990	60	Necessity
M. H. F.	Cardiac hypertrophy	Recumbent Sitting	39 32	520 660	480 580	1000 1240	190	Necessity

* The terms "complementary air" and "supplementary air" are used to denote the greatest amounts of air which can be inspired and expired respectively, from the mid-position of the lungs.

than in the recumbent in the normal subjects and in the patient who had no orthopnea, was moderately greater in the two subjects with orthopnea of choice, and was markedly greater in the subjects with severe orthopnea. The supplementary air was greater in all subjects in the sitting posture, the degree of increase being about the same in the patients as in the normal subjects. The complementary air was less in the sitting than in the recumbent posture in the normal subjects and in the patient with no orthopnea. In the orthopneic patients the complementary air was unchanged or greater in the sitting posture. The shift in the mid-position of the lungs on change of posture was usually greater than the change in vital capacity in the normal subjects, but the reverse was true in the orthopneic patients. Consequently the latter usually had greater complementary air in the sitting posture and the former had more "room to breathe" in the recumbent position. These observations may throw some light on the facts that normal subjects may breathe slower when recumbent and that orthopneic individuals are likely to breathe slower when sitting.

C. *The relations of the position of the head and of venous pressure to orthopnea*

Ernstene and Blumgart (1930) observed that in orthopneic patients elevation of the head without raising the body was often followed by distinct diminution in respiratory discomfort. They concluded that this relief was to be attributed to increased cerebral blood flow due in turn to diminished venous pressure. The data already presented (Table 1) indicate that the blood flow through the head is not changed by posture in patients with orthopnea. However, although we disagree with their conclusion, we have confirmed their observation (Table 4). In seven of eight observations on seven patients the ventilation was less with the head flexed than with it extended. As significant changes in respiratory rate and in vital capacity were not observed these data are omitted from the table. Six of the seven patients said that they were somewhat more comfortable with the head flexed although in most instances the difference was not striking.

In order to determine whether the increase in venous pressure produced by extending the head was in itself responsible for the increased respiratory discomfort felt by the patients further observations were

TABLE 4

Ventilation in recumbent patients with the head extended and with the head flexed

Subject	Chief diagnosis	Date	Degree of orthopnea	Position of head	Ventilation per minute per meter	Degree of subjective relief produced by flexing head
		1931			*liters*	
D. W.	Hypertension	January 15	Necessity	Extended	6.56	
				Flexed	5.94	Slight
A. B.	Hypertension	January 15	Choice	Extended	5.24	
				Flexed	4.54	Slight
W. M. J.	Syphilitic aortic insufficiency	January 14	Necessity	Extended	4.62	
				Flexed	3.82	Moderate
		January 17	Necessity	Extended	4.76	
				Flexed	4.68	Slight
C. M.	Hypertension Emphysema	January 19	Necessity	Extended	5.12	
				Flexed	4.76	Slight
J. L.	Asthma Emphysema Tuberculosis	January 14	Necessity	Extended	4.64	
				Flexed	4.06	Slight
J. D.	Mitral stenosis Aortic insufficiency	January 14	Choice	Extended	6.68	
				Flexed	5.90	None
W. C.	Hypertension	January 10	Choice	Extended	3.58	
				Flexed	3.58	None

made on both normals and patients. A blood pressure cuff was put around the neck of the recumbent subject. The ventilation was measured for 3 minutes with no pressure, and successively after the cuff had been inflated to 15 mm. Hg with the patients and to 15, 25 and 35 mm. with the normals. The head was then flexed and the measurements at various pressures repeated. Finally the subjects were propped up in the sitting position and the same observations were made.

The results for the normals are shown in Figure 2, each point being the average value for four to six measurements. In each position increase in the pressure caused some increase in ventilation. At all de-

grees of pressure the ventilation was least in the horizontal position with the head flexed and most in the sitting position, the values for the reclining position with the head extended being intermediate. However, with moderate pressure (15 and 25 mm.) the increase over the

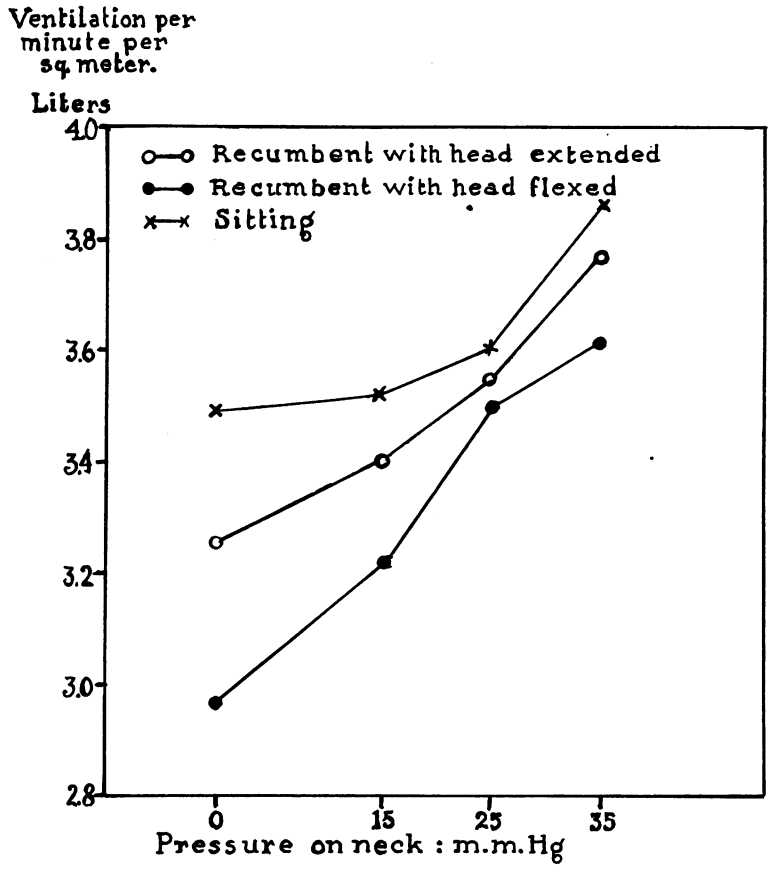

FIG. 2

Shows relationship between pressure on neck and ventilation in various positions. All subjects normal.

value with no pressure was less in the sitting than in the two recumbent positions.

The subjective sensations of the normal subjects are of especial interest. In addition to the local sense of pressure on the neck which

was incidental to the procedure, all of them experienced an uncomfortable feeling of fullness in the head. The degree of this sensation was naturally proportional to the height of the pressure, but each of the subjects noted that for any given pressure he was distinctly less uncomfortable with the head flexed than with the head extended, and was least uncomfortable in the sitting position. *None of the subjects felt any shortness of breath.* If rise in venous pressure *per se* is an important cause of cardiac dyspnea they should have been quite short of breath because the venous pressures in the head were decidedly higher than those ordinarily found in patients with congestive heart failure.

Observations were made on the effect of artificially increasing the venous pressure to 15 mm. Hg in five patients. Three of them had had congestive failure with orthopnea in the past but did not have orthopnea at the time the observations were made. In the recumbent posture their ventilation was measured with and without putting pressure on the neck. Significant increase in ventilation was not observed (Table 5). Subjective dyspnea did not occur. The other two patients had severe orthopnea and were studied in the upright position. Again significant changes in ventilation and in subjective respiratory distress did not take place when pressure was applied to the neck. These observations seem to be fairly conclusive proof that increase in venous pressure is not the major factor in the production of orthopnea.

These observations seem to us to clarify the significance of the position of the head and of venous pressure in the production of orthopnea. Regardless of the height of venous pressure recumbent individuals feel somewhat uncomfortable with the head extended (most people prefer to sleep on a pillow). The higher the venous pressure the greater is the discomfort in this position.

Distress of any kind tends to cause increase in breathing. This slight increase in ventilation causes no discomfort in normal subjects because of their large respiratory reserve (vital capacity). The orthopneic patient is already calling on his reserves and the slight increase in ventilation which comes when the head is extended causes further respiratory distress. It should be noted that the relative difference in ventilation on flexing the head was about the same (ten per cent) in the normal subjects and in the patients. However, the actual decrease in ventilation on flexing the head was almost twice as much in the patients.

TABLE 5

The effect of pressure on the neck on ventilation

Subject	Diagnosis	Degree of orthopnea	Position	Pressure on neck	Ventilation per minute per square meter	Degree of respiratory distress
				mm. Hg	*liters*	
F. J.	Bronchitis Cardiac hypertrophy	None	Recumbent	0 16	4.03 3.57	None
A. C.	Hypertension Cardiac hypertrophy	None	Recumbent	0 16	4.40 4.47	None
L. C.	Hypertension Cardiac hypertrophy	None	Recumbent	0 16	3.77 4.30	None
W. C.	Hypertension Cardiac hypertrophy	Necessity	Sitting	0 15	4.89 4.60	Slightly more dyspneic with pressure on neck
Al. C.	Syphilitic aortic insufficiency	Necessity	Sitting	0 15	7.26 7.73	Unchanged

It is possible that changes in venous pressure or in position of the head or neck may, through reflexes, have some influence on breathing. It is conceivable that alterations of the blood pressure in the carotid sinus is responsible, but of this we have no direct evidence.

SUMMARY

Orthopnea is a complex phenomenon. A number of factors may play a rôle in its production. An attempt has been made to evaluate their relative importance in this study and the following conclusions have been reached:

1. *Decreased cerebral blood flow in the recumbent as compared to the sitting posture* apparently does not occur. The amount of oxygen taken out of the blood passing through the brain is, in orthopneic patients, almost exactly the same in the two positions and, although figures from different individuals show wide variations, the average figure is ap-

proximately the same as that found in normal subjects. Hence, changes in cerebral blood flow are probably of no significance in the production of cardiac dyspnea either in the sitting or recumbent postures.

2. *Oxygen saturation and acid-base condition.* The arterial saturation often is somewhat greater in the sitting than in the horizontal position. The degree of change is frequently too small to be of great importance and even in recumbent orthopneic patients the arterial saturation is usually within normal limits. The carbon dioxide content of the arterial and internal jugular blood is usually normal and is relatively unaffected by posture. In both positions the carbon dioxide tension and hydrogen ion concentration of blood entering and leaving the brain are usually lower than the average normal and in most cases the values are not changed beyond the limits of error of the methods, by change of posture. In certain cases, particularly those with asthma, the pH may be low and the carbon dioxide tension high in the recumbent posture, and in such patients sitting up is followed by a shift toward alkalinity in these functions. Ordinarily such alterations in pH and CO_2 tension as are found in both positions are to be regarded as effects rather than causes of dyspnea. It is therefore evident that in the majority of patients with orthopnea due to congestive heart failure, deficient aeration of the blood in the recumbent posture is either of no significance or of only slight importance.

3. *The position of the head* is of some significance in almost all orthopneic patients. The relief produced by flexing the head is not due to change in cerebral flow.

4. *Increased respiratory rate* in the recumbent posture is also of importance in the production of severe orthopnea in certain patients.

The cause of the increased respiratory rate in patients with cardiac failure and the reason for the further increase, in some cases, on assuming the horizontal posture is not yet entirely known. It is not due to changes in the oxygenation or acid-base condition of the blood.

5. *Diminution in vital capacity.* In patients with advanced cardiac failure a diminution is found invariably. In the sitting posture the patients respiratory reserve is much decreased, i.e., he is near the threshold of dyspnea. On lying down there follows a further decrease in vital capacity which is not only relatively but also usually actually

greater in patients with congestive failure than in normal individuals. In our patients the average increase in vital capacity in the sitting as compared to the horizontal position was 460 cubic centimeters or twenty-seven per cent of the recumbent value. The fraction of the vital capacity used per breath is therefore greater in the recumbent posture and this is, in large measure, responsible for their distress on lying down. Changes in vital capacity and lung volume are to be regarded as the most important causes of orthopnea.

These conclusions are in agreement with those of Peabody (1916–17) and his co-workers in indicating the importance of diminished vital capacity in cardiac dyspnea in general and with those of Christie and Beams, and of Field and Bock (1925) in regard to the significance of changes in vital capacity in the production of orthopnea.

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