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Research Article

ORTHOPNOEA AND THE EFFECT OF POSTURE UPON THE RATE OF BLOOD FLOW

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Lindhard (1) has noted that, of seven subjects, three, all of them females, had a slower rate of blood flow in the sitting than in the recumbent posture while the four male subjects showed practically no change. As far as we know these are the only direct observations in the literature concerning the effect of posture on blood flow.

We have made thirteen determinations of the circulatory rate with the subjects sitting and nine determinations with the subjects standing, each one compared with a preceding determination made while the subject was recumbent. A rest period of about an hour preceded the first determination in each experiment. Change of position was accompanied by as little exertion as possible and a further rest period of fifteen to twenty minutes preceded each determination in the new position. The method used is a gasometric one, based on the Fick principle, which we have previously described (2). The subjects were young, healthy, adult males except the last two who were females and G. R. who has a rheumatic cardiac lesion which is well compensated. None of the subjects were athletic.

Our results, shown in table 1 are even more striking than those of Lindhard.

In no case did we fail to observe a diminution in the rate of blood flow on changing from the recumbent to the sitting position. Only once was the reduction less than 500 cc. per minute, while another observation on the same individual showed a greater change.

The slowing of the circulation was more marked in the standing than in the sitting position. The average rate of blood flow while sitting was 76 per cent and while standing it was 50 per cent of the

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rate while reclining. Inasmuch as the pulse rate increased from an average of 63 per minute in the reclining position to 65 per minute in the sitting position, and 90 per minute in the standing position, the output per beat was tremendously diminished in the upright posture. While standing it amounted to only 35 per cent (on the average) of

TABLE 1

Subject	Lying			Sitting*			Standing		
	Pulse	Circulation per minute	Output per beat	Pulse	Circulation per minute	Output per beat	Pulse	Circulation per minute	Output per beat
	per minute	*liters*	*cc.*	*per minute*	*liters*	*cc.*	*per minute*	*liters*	*cc.*
A. V. B.	66	5.11	77				88	3.95	45
	65	6.49	100	68	5.73	84			
	68	6.30	92.5	68	5.80	85	78	4.46	57
H. F.	73	9.87	136				95	4.67	49
	72	9.60	133	80	7.29	91			
	68	8.66	131	70	8.42	120	89	3.90	44
A. C. R.	60	8.90	148	64	3.75	59	83	4.12	50
S. A. O.	54	9.73	180	66	7.08	107	79	4.42	56
	58	6.52	112	59	5.81	98.5			
J. R. L.	56	9.04	161	54	4.92	91	76	3.12	41
W. A. Mc.	55	8.12	148	57	5.66	99			
K. M. M.	58	6.69	115	58	5.28	91	105	3.41	32.5
G. R.	62	10.90	176	56	8.07	144			
L. G.	62	8.27	133	66	7.40	72			
M. E. M.	62	6.42	103.5	80	4.21	53.5	115	4.05	35
Average	63	8.04	127.6	65	6.11	94.0	90	4.01	44.6
Per cent of reclining value				103	76	74	143	50	35

* The values obtained for the blood-flow in the sitting position are not unlike those published by Burwell and Robinson in volume one of this Journal. Their determinations were made upon subjects at rest in a reclining position in a hospital wheel chair.

that while reclining. This is also indicated by the blood pressure findings. After the subject had been standing for a time the pulse pressures were greatly diminished, often to 5 or 10 mm. of mercury, and sometimes the auscultatory vascular sounds were almost inaudible.

As a part of a study of circulatory and respiratory conditions in

college girls, many of whom were especially athletic, Miss Abby H. Turner (3) has made similar determinations in the laboratory of Physiology of the Harvard School of Public Health, using our method. Of 32 determinations of blood flow in the sitting position, 8 showed a rise, 7 little or no change and 17 a distinct fall from the resting volume. Of 19 determinations made in the standing position, 1 showed a larger volume of blood flow than in the reclining position, 4 showed practically no change, and 14 a smaller volume flow.

Y. Henderson and Haggard (4) have recently repeated these experiments by a new method and have obtained quite similar results. As the phenomenon has thus been observed in four different laboratories by three different methods, there can hardly be any question as to its occurrence. Its cause and its significance, on the other hand, are interesting subjects for speculation.

DISCUSSION

The obvious explanation is to attribute the change in blood flow to an effect of gravity which, in the upright position, produces a stasis of the circulation in the legs and splanchnic area which results, consequently, in a diminished supply of blood to the heart. It is well known that holding a rabbit suspended by the ears will in a short time result in syncope and, after a variable time, in death. Soldiers sometimes faint during long periods of standing at attention. We have noted that while a subject was standing during an experiment the calves of his legs became indurated and brawny and increased in circumference about 1.5 cms.

L. Hill (5) and Hill and Barnard (6) have studied the effect of posture on the arterial and venous blood pressures of animals. They found that the normal dog was able to compensate, in part, for the effect of gravity and to maintain a circulation in the vertical position with feet down although with a considerable drop in the blood pressure of the arterial system and of the superior vena cava. When the splanchnic vaso-motor control and the respirations were inhibited, however, assumption of the vertical feet down position was followed by a drop in the arterial blood pressure to zero and, in a short time, in death of the animal.

Hill found that the vertical position had much less effect on the blood pressure in monkeys than in dogs, and concluded that their compensatory mechanisms were more highly developed. Our experiments might be taken to indicate that even man is not completely adjusted to the upright position. Or perhaps one of the adjustments consists in a rate of blood flow which, in the reclining position, is so much more rapid than the needs for gas transport demand, that the slower flow in the upright position is still within the limits of safety so that no difficulty usually develops.

The diminution of the arterial pulse pressure in the upright position has been noted. We have made a considerable number of determinations of the blood pressures in arm and hand veins with the subjects in the different positions. Both the indirect method of Hooker (7) and a direct method with a needle inserted into a vein and attached to a manometer have been used. The results have been indeterminate. No great changes in venous blood pressures have been found associated with change in position. Nor has the direction of variation been constant. More often there has been some rise in the venous blood pressure after assuming the upright position. Barach and Marks (8) have reported similar changes in arterial and venous blood pressures associated with change in position.

A review of previously reported studies, however, indicates that this need be neither very surprising nor disturbing. Kroetz (9) after a long series of determinations in patients came to the conclusion that there were too many unknown factors influencing the venous blood pressure to attach much significance to it in relation to the general circulation. Wiggers (10) found that, although the return flow of blood was reduced in the early stages of shock, the venous blood pressure remained unchanged or increased and has stated (11) that "as long as the return flow of blood is not reduced too greatly the mean auricular pressure may be maintained by compensatory mechanisms." The association of high venous blood pressures with diminished rates of blood flow, which we have observed to occur in the upright position in man, is a demonstration of such compensatory mechanisms. There is a certain amount of evidence of venomotor activity. The subject needs further investigation. Cardiac tonus might also be a factor

in maintaining the venous blood pressure but the existence of such a tonus has not been proved.

The observations herein reported have led us to a consideration of the present conceptions concerning orthopnoea and the possible relationship between the two phenomena—slower blood flow and more comfortable breathing in the upright position.

L. Hill (5) in 1895 expressed an idea which for a time we thought was original with us. He said "The position selected by patients suffering from lung or heart disease can be explained, for it is manifest that the upright position will afford the greatest relief by diminishing pulmonary congestion through the retention of a large quantity of blood in the splanchnic area." Since that time there have been numerous studies of dyspnoea and orthopnoea yet the statement has frequently been made that there is no satisfactory explanation for orthopnoea. We have seen no further reference to Hill's suggestion.

Concerning the details of the production of dyspnoea itself there may still be some debate. The resting metabolism is frequently found to be increased in dyspnoeic cardiac patients as shown by Peabody, Meyer and DuBois (12), and by Peabody, Wentworth and Barker (13). This increases the demand for respiratory exchange. The respiratory exchange is carried on less efficiently in such patients than in normal persons. They need to breathe a larger volume of air per minute in order to accomplish the same carbon-dioxide elimination and oxygen absorption. Siebeck (14) argued that this was due to an inadequate mixture of air in the lungs and an increase in the functional dead space. Peters and Barr (15) thought that there is an increased tension difference between the gases in the alveolar air and the arterial blood because of a diminished diffusion capacity of the lungs. Peabody, Sturgis, Barker and Read (16) unlike Siebeck and Peters and Barr, found that in their cases, although the total ventilation was considerably increased, because of the fact that breathing was more rapid and shallow than normal, the effective ventilation (the total ventilation minus the total dead space air) was not increased out of proportion to the metabolism. Consequently they did not need to invoke either of the above mentioned mechanisms. Their subjects were not as dyspnoeic as those of the

previous workers and it may well be that inefficient gas exchange in the lungs is a late development.

Finally the ability of the dyspnoeic cardiac patient to increase the volume of his ventilation is diminished. It has been known for a long time that the vital capacity of cardiac patients is reduced. Peabody and Wentworth (17) demonstrated that the amount of the reduction is closely related to the degree of dyspnoea that the patient suffers. Sturgis, Peabody, Hall, and Fremont-Smith (18) have given the relationship a more exact expression by showing that the maximum ventilation both in normal subjects and in cardiac patients is obtained by increasing the respiratory rate to about thirty-five per minute and the depth of respiration to about one-third of the vital capacity.

Behind all of these factors, however, it seems quite certain, although perhaps not absolutely proven, that the fundamental cause of dyspnoea is congestion of blood in the pulmonary circuit. Basch in 1891 (19) proposed the idea that the increased turgidity of the congested pulmonary vessels diminished the flexibility of the lungs. This is supported by the researches of Lundsgaard (20) who found that the earliest change in the lung volumes of cardiac patients was an increase in the residual air. This occurred before there was any change in the total capacity and was therefore the cause of the early decrease in the vital capacity of the lungs. Peabody, Sturgis, Barker, and Read (16) found that compensated cardiac patients, with little reduction in their vital capacities, had increased respiratory volumes per unit of metabolism because of more rapid, shallow breathing, with a consequent increase in the proportion of dead space air to the total expired air. This they suggest is possibly explained by an earlier excitation of the Hering-Breuer reflex due to the increased rigidity of the lungs.

Greater degrees of pulmonary congestion cause encroachment upon the air space in the lungs. Romanoff (21) found that following increases of pressure in the pulmonary circuit the lungs expanded if the pleurae were opened, but that if such expansion were restricted by intact pleurae the alveolar air space was contracted. Drinker, Peabody and Blumgart (22) and Drinker and Agassiz (23), found that following compression of the pulmonary veins, in their preparation with intact pleurae, there was an increased blood pressure in the

pulmonary artery, while a diminished proportion of air from a constant delivery respiration pump entered the lungs and an increased proportion passed through an overflow tube. Such observations are thought to have a direct bearing on the diminished vital capacities of cardiac patients.

Bohr in 1907 (24) found that the vital capacity is less in the reclining position than it is while standing. He reported only a small series of observations, and was chiefly interested in changes in the mid-capacity. Christie and Beams (25) have recently determined that the average vital capacity of 290 normal people was 5.5 per cent less while lying than while sitting. They also found (26) that in 19 patients with diminished vital capacities but without orthopnoea this difference was 4.7 per cent; in 14 patients with "orthopnoea of choice" it was 12.5 per cent; while in 9 patients with "orthopnea of necessity" it was 26.7 per cent. There was not only this very great increase in the relative difference between the vital capacities, while lying and sitting in the latter group, but also an increase in the absolute difference of about 50 per cent. Christie and Beams believe that this effect of posture on vital capacity is the primary factor in the production of orthopnea.

It remains to consider the fundamental cause that is responsible for this effect. For years the chief explanation for orthopnea has been a lowering of the diaphragm and a supposed increase in the capacity of the chest in the sitting posture. With the descent of the ribs and the narrowing of the intercostal angle that occurs it is questionable if the capacity of the chest is not actually diminished in this position. At any rate, even if such an anatomical relationship were responsible for part of the difference between the vital capacities, lying and sitting, it would not explain the increase in this difference in patients suffering from heart disease. There must be some other factor which is peculiar to heart disease and which is susceptible to the influence of change of position.

It has been argued that the sitting posture permits better use of the muscles of respiration. Such an explanation has never been accepted as satisfactory. Moreover, Peabody and Sturgis (27) have shown that even extreme general weakness does not greatly diminish the vital capacity. Also frequently repeated determinations of the vital ca-

pacity in cardiac patients, a procedure likely to cause considerable fatigue, does not result in its further reduction.

It seems much more reasonable to regard as important the condition which is primarily responsible for the reduced capacity of the lungs, that is, the pulmonary circulation. Many researches, too numerous to analyze here, have shown the great susceptibility of the pulmonary blood pressure to various influences. Plumier (28) demonstrated that compression of the inferior vena cava causes a reduction in pulmonary arterial pressure. Pressure on the abdomen by increasing the blood supply to the heart raises it. Cloetta and Staubli (29) found that compression of the aorta or a quick infusion of 15 cc. of salt solution into a cat, not only caused a rise in pulmonary arterial pressure but an increase in the volume of the lung in a plethysmograph, which if the lung were encased in a non-expansive box, would greatly diminish the alveolar air space. Fuhner and Starling (30) found that increased venous supply and increased peripheral resistance raised the pulmonary arterial pressure in their heart-lung preparations. Drinker and Agassiz (23) produced similar rises by compression of the pulmonary veins.

The diminished output of the heart which we have demonstrated occurs in the upright position is admirably adapted to reduce the congestion in the lungs, to increase the vital capacity and to make breathing more comfortable. It should give relief in much the same way as does venesection. Instead of the total volume of the blood being reduced in volume, a part of it stagnates in the dependent parts; a smaller amount is returned to the heart; the output of the right heart is diminished; this smaller amount of blood should be more readily dealt with by the left heart and pumped on into the systemic circulation; and the congestion in the lungs should be relieved. A degree of cardiac rest also results from the reduced output of blood.

It will undoubtedly appear strange at first sight that a dyspneic cardiac patient, with a circulation which is already slower than normal, should be relieved by still further slowing of the circulation. It may be pointed out that the normal resting blood flow in man is much faster than the needs for gas transport demand so that there is a wide margin of safety. The tissues, aside from those of the nervous system, stand the anoxemia and the relatively slight increase in

carbon-dioxide tension, which would result from a slow circulation, comparatively well. The nervous system, on the other hand, has such a large blood supply in proportion to its metabolism that its blood is never subject to very great venous change. This being so, a slower blood flow does not cause an increase in the respiratory stimulus as long as the blood does not leave the lungs less well ventilated. The respiratory center has such a large blood supply that the capillary blood which controls it is practically arterial blood. Slower blood flow should not therefore impair the ventilation of the arterial blood. A small volume of blood that has been highly utilized, can be as readily freed of carbon dioxide, and as readily oxygenated by the respiratory mechanism as a larger volume of blood that has been less completely utilized. The respiratory demand is a function of the metabolism rather than of the circulation.

Clinical confirmation of this fact is frequently observed. Fainting rather than dyspnea is the reaction to a diminished blood flow. Mediastinal lesions, that do not involve the pulmonary circulation, may cause marked venous engorgement, cyanosis and stasis of the circulation in the upper part of the body without being necessarily accompanied by any considerable degree of dyspnea.

Throughout this paper the argument has been presented from the standpoint of the cardiac patient. The discussion also applies to persons who have dyspnea and orthopnea because of diminution of the vital capacity due to other causes than heart disease. As Christie and Beams (26) pointed out even the 5.5 per cent increase in vital capacity which normal subjects gained by changing from the lying to the sitting position may mean the difference between extreme embarrassment of the respiration and comparative comfort. It is also quite possible that lesions other than those of the heart, which reduce vital capacity, may also cause pulmonary congestion that may be relieved in the same manner.

SUMMARY

Data showing a diminution in the rate of blood flow in the upright position, thought to be due to an effect of gravity, impeding the return of venous blood to the heart, have been presented.

The manner in which the diminished blood flow in the upright

position may reduce dyspnea by relieving the congestion in the pulmonary circulation, has been discussed.

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