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Research Article

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# THE EFFECT OF POSTURE ON PULMONARY CAPILLARY BLOOD FLOW IN MAN *

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The alterations in cardiac output resulting from changes in posture are accompanied by shifts in blood volume and changes in venous return (1–8). Recent radioisotope studies have shown that, in addition to these general effects, posture has important influences on the distribution of blood flow to different parts of the lungs (9). The apexes receive relatively little blood flow when a person is upright, but the flow is more uniformly distributed when he is horizontal. Since pulmonary capillary blood flow had been studied only in seated subjects, we studied the effects on it of changes in posture (10). By minimizing the shifts of blood volume ordinarily associated with changes in posture, the effect of postural change per se on cardiac output, stroke volume, and instantaneous pulmonary capillary blood flow was determined.

## METHODS

Six normal men, aged 27 to 39, were studied 1 to 2 hours after they had eaten lunch. Stroke volume and instantaneous pulmonary capillary blood flow were measured by the technique of Wasserman and Comroe (11). A multichannel oscillographic recorder provided simultaneous tracings of $N_2O$ absorption, ECG, $N_2O$ concentration, and thoracic volume (by pneumographs). The mechanical properties and the response characteristics of the system have been reported elsewhere (11). Brachial arterial blood pressure was determined by auscultation with an aneroid sphygmomanometer.

The experimental procedure consisted of measuring stroke volume, instantaneous pulmonary capillary blood flow, pulse rate, and blood pressure of the subjects on a tilt table in the following sequence of positions: *1)* horizontal; *2)* upright, at an angle of 60°; *3)* upright, with pneumatic cuffs around the thighs inflated to a pressure of 60 to 70 mm Hg [previous investigations (6, 12) have shown that this procedure prevents shifts in blood volume caused by changes in posture]; *4)* horizontal, with the pneumatic cuffs still inflated; *5)* horizontal, after the cuffs were deflated.

Each position was maintained for 30 minutes, and measurements were made after the initial 10 minutes to ensure that the pulse rate and blood pressure had reached a steady state.

## RESULTS

*Stroke volume and cardiac output.* When the subjects were tilted from the horizontal to the upright position, the stroke volume and cardiac output always fell to 60 to 76 per cent of the control level (Table I), and remained low after inflation of the pneumatic cuffs. Although limb volume was not measured directly, the cuffs probably did not trap blood in the legs, since inflating the cuffs had no appreciable hemodynamic effect in any subject. Two subjects had a moderate degree of ankle edema. In the horizontal position with the cuffs still inflated, stroke volume was unchanged or slightly increased, but when the cuffs were deflated it increased to the control level.

The cuffs were deflated while the subject held his breath at the resting lung volume, with his airway opened to the Krogh spirometer. As shown in Figure 1, within 2 or 3 seconds after deflating the cuffs, air was forced out of the lungs into the spirometer, and the pneumograph recorded a simultaneous increase in chest volume. (This can be interpreted only as representing an increase in thoracic blood volume.) In the example shown, the thoracic blood volume increased about 200 ml after deflation of the cuffs. Close inspection of both the pneumograph and spirometer tracings reveals increased amplitude of the cardiogenic oscillations after the shift of blood into the thorax, presumably because of an increase in stroke volume.

The effect of posture and distribution of blood volume on pulse rate and blood pressure is shown in Figure 2. In three subjects (A.N., R.Y., P.S.)

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pulse rate changed only slightly from control values, once a steady state had been achieved. In the other three it increased when they were upright and decreased when they were tilted back. Since there was little change in blood pressure and a fall in cardiac output in all subjects when they were upright, total peripheral vascular resistance must have increased appreciably in this position.

*Instantaneous pulmonary capillary blood flow.* The method we used to measure $N_2O$ uptake re-

TABLE I

*The effect of posture and distribution of blood volume on cardiac and stroke indexes in six normal men* *

| Determination | Horizontal | Upright (60°) | Upright (60°), cuffs infl. | Horizontal, cuffs infl. | Horizontal, cuffs defl. |
|---|---|---|---|---|---|
| A.N., age 27, 180 cm, 81.0 kg, 2.02 $m^2$ | | | | | |
| C.I. | 3.08 | 1.88 | 2.04 | 1.82 | 2.80 |
| | 2.95–3.18 | 1.60–2.20 | 1.93–2.28 | 1.60–1.95 | 2.58–2.90 |
| S.I. | 50 | 30 | 32 | 30 | 46 |
| | 47–54 | 28–36 | 30–36 | 27–33 | 43–49 |
| H.R. | 62 | 63 | 64 | 60 | 62 |
| | 60–65 | 60–65 | 63–64 | 60–61 | 60–64 |
| R.Y., age 31, 187 cm, 79.5 kg, 2.00 $m^2$ | | | | | |
| C.I. | 2.36 | 1.55 | 1.59 | 1.46 | 2.45 |
| | 2.25–2.45 | 1.55–1.55 | 1.59–1.60 | 1.42–1.49 | 2.23–2.68 |
| S.I. | 40 | 26 | 26 | 26 | 40 |
| | 39–41 | 25–27 | 26–27 | 25–27 | 35–44 |
| H.R. | 59 | 59 | 61 | 56 | 62 |
| | 58–60 | 57–61 | 59–62 | 55–58 | 60–64 |
| P.S., age 39, 185 cm, 84.0 kg, 2.10 $m^2$ | | | | | |
| C.I. | 3.63 | | 2.07 | 2.34† | 3.11 |
| | 3.38–4.09 | | 1.92–2.21 | | 3.00–3.21 |
| S.I. | 52 | | 30 | 35† | 48 |
| | 49–58 | | 29–31 | | 45–49 |
| H.R. | 70 | | 69 | 66† | 65 |
| | 69–71 | | 67–60 | | 67–69 |
| J.B., age 37, 187 cm, 86.5 kg, 2.10 $m^2$ | | | | | |
| C.I. | 4.49 | 2.83 | 2.76 | 3.07 | 3.65 |
| | 4.33–4.65 | 2.48–3.18 | 2.38–3.14 | 3.00–3.14 | 3.64–3.68 |
| S.I. | 58 | 35 | 32 | 44 | 61 |
| | 58–59 | 29–45 | 29–36 | 43–45 | 60–62 |
| H.R. | 77 | 84 | 86 | 68 | 57 |
| | 75–79 | 73–100 | 84–88 | 67–69 | 55–58 |
| D.W., age 34, 175 cm, 71.0 kg, 1.85 $m^3$ | | | | | |
| C.I. | 3.55 | 2.56 | 2.13 | 2.24 | 3.33† |
| | 3.35–3.72 | 2.52–2.57 | 1.76–2.46 | 1.93–2.52 | |
| S.I. | 51 | 32 | 28 | 33 | 50† |
| | 47–55 | 31–35 | 25–27 | 27–38 | |
| H.R. | 70 | 79 | 75 | 68 | 67 |
| | 68–71 | 78–79 | 71–79 | 66–69 | 66–69 |
| D.D., age 36, 172 cm, 74.0 kg, 1.83 $m^2$ | | | | | |
| C.I. | 2.53 | 1.93 | 1.79 | 1.90 | 2.50 |
| | 2.50–2.64 | 1.82–2.05 | 1.64–1.94 | 1.70–2.10 | 2.45–2.66 |
| S.I. | 38 | 26 | 24 | 27 | 40 |
| | 36–40 | 26–27 | 22–27 | 24–30 | 39–40 |
| H.R. | 67 | 74 | 75 | 71 | 63 |
| | 65–68 | 69–79 | 73–77 | 70–72 | 62–64 |
| Mean percentage of control value | | | | | |
| C.I. | 100 | 65 | 62 | 64 | 91 |
| S.I. | 100 | 63 | 60 | 67 | 95 |

* Values shown are mean and range of 3 to 5 determinations in each situation unless otherwise indicated. C.I. = cardiac index (L/min/$m^2$); S.I. = stroke index (ml/$m^2$); H.R. = heart rate (cycles/min).

† Single determination.

quires the subtraction of total gas absorption after a control breath of air from that after a breath of $N_2O$. This requires use of cardiac cycles of identical duration to calculate instantaneous $N_2O$ uptake (11). In two subjects instantaneous flow could not be measured in all positions because of variability of pulse rate. The results in the other four are illustrated in Figure 3. Blood flow through the pulmonary capillaries was clearly pulsatile in all experimental positions. The flow curves pass through a minimum, frequently zero, at the time of inscription of the R wave of the ECG. Peak flow occurred just after the T wave. In some persons (A.N. in this study), tracings indicate back flow in the pulmonary capillaries. This may be an artifact, since it occurs when the tracings are changing direction abruptly and the

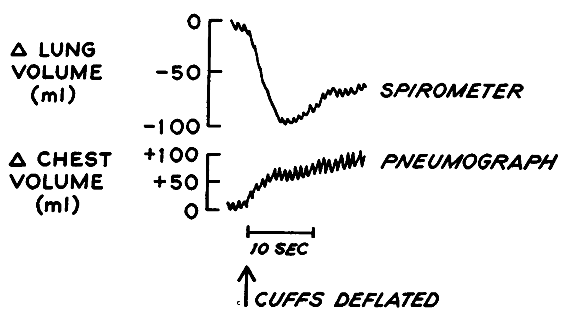


FIG. 1. INCREASE IN CHEST VOLUME AND DECREASE IN LUNG VOLUME AFTER DEFLATION OF PNEUMATIC CUFFS.

subtraction procedure is more open to error than it is later in the cardiac cycle. The fact that it appears regularly in some persons, however, suggests that it is real. Even if back flow occurs, one would expect only minute amounts of $N_2O$ (1 per cent of mean uptake) to be added to the alveolar gas, because the last blood to leave the pulmonary capillaries (and the first to flow back) would have virtually the same tension as alveolar gas. An alternative possibility exists. If $N_2O$ tension equilibrates more slowly between alveolar gas and pulmonary parenchymal tissue than it does between alveolar gas and pulmonary capillary blood, almost 8 per cent of the total uptake of $N_2O$ may enter the alveoli from the parenchymal tissue when blood flow falls to zero (11).

Although the pattern of flow was similar in all positions, the peak flow was reduced in the upright. In the horizontal, with pneumatic cuffs inflated,

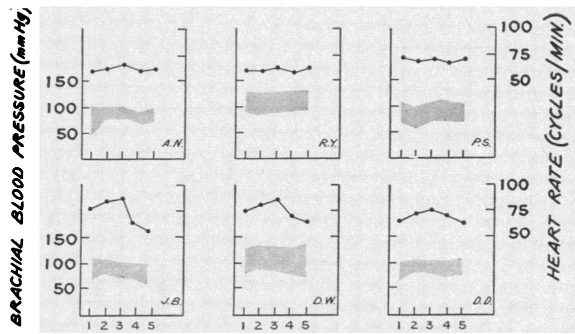


FIG. 2. EFFECT OF POSTURE AND DISTRIBUTION OF BLOOD VOLUME ON PULSE RATE AND BLOOD PRESSURE. Abscissa numbers correspond to sequence of positions described in Methods.

the peak flow remained low but increased to the control value when the cuffs were deflated. The changes in amplitude of the flow pulse with change in posture are therefore related to the changes in stroke volume (see above). This correlation between peak flow and stroke volume is shown in Figure 4.

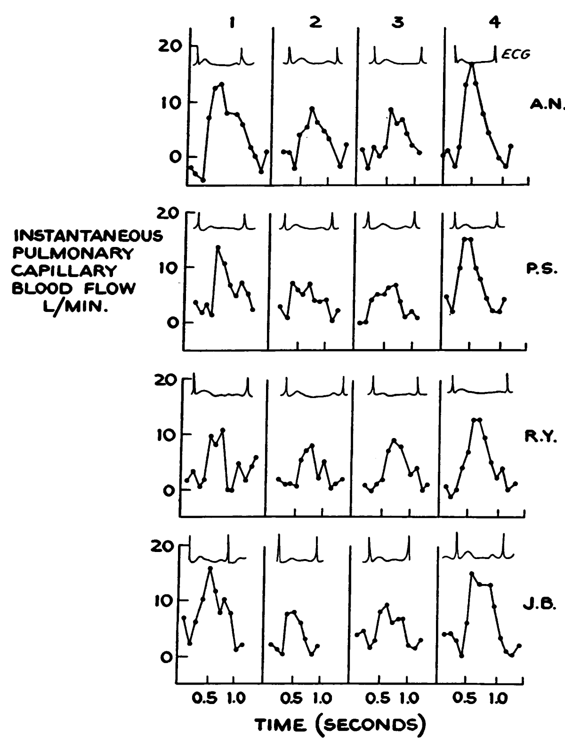


FIG. 3. EFFECT OF POSTURE AND DISTRIBUTION OF BLOOD VOLUME ON INSTANTANEOUS PULMONARY CAPILLARY BLOOD FLOW. 1) Horizontal; 2) upright; 3) horizontal, cuffs inflated; 4) horizontal, cuffs deflated.

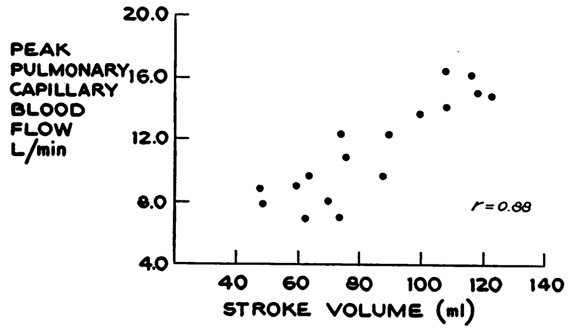


FIG. 4. RELATIONSHIP BETWEEN PEAK PULMONARY CAPILLARY BLOOD FLOW AND STROKE VOLUME.

## DISCUSSION

Theoretically, the uptake of $N_2O$ from the lungs is proportional to the blood flow through the pulmonary capillaries.[1] In normal persons, in whom there is only a small "physiological" right-to-left shunt, the $N_2O$ and other foreign-gas methods yield values which closely approximate the total cardiac output as measured by the direct Fick and indicator dilution techniques (10, 11, 13). The use of a foreign-gas method has the advantage of not requiring cardiac catheterization, injections, or chemical analysis of blood. Certain other considerations make foreign-gas methods especially advantageous for the study of physiological phenomena in resting man. We have found, for example, that stroke volume measurements are less variable than are those obtained by the direct Fick or dye dilution methods. This is illustrated in Table II, in which an analysis of the variability of duplicate determinations of cardiac output and stroke volume, reported by Holmgren and Pernow who used the direct Fick method, is compared with a similar analysis of duplicate determinations made during the present study (14). We have attributed the reduced variability with the $N_2O$ method to absence of the psychic effects of pain and apprehension (2, 15). The chief value of this method, however, is that it permits an estimate of instantaneous flow in the pulmonary capillaries.

Our data demonstrate the well known changes in stroke volume and cardiac output that accompany changes in posture (1–8). These changes may be regarded as passive phenomena resulting from alterations in the distribution of blood volume. It has been shown, for example, that a change from lying to sitting or standing increases venous pressure and causes vascular dilatation in the legs and perhaps elsewhere, depending on the compliance of the vessels (16, 17).

When an adult stands, up to 500 ml of blood may pool in his legs (6). These findings have led investigators to conclude that stroke volume and cardiac output are reduced in the upright posture because of a reduction in the thoracic blood volume and impaired diastolic filling of the heart (4). This is supported by studies that show a reduction in thoracic blood volume and heart size in the upright posture (18, 19). Other factors that might be partly responsible for the postural changes in cardiac output and stroke volume have not previously been assessed. These include alterations in the position of the heart and great vessels, reorientation of the vestibular apparatus, and proprioceptive influences from the periphery. Our studies showed that changes in stroke volume and cardiac output that accompany changes in position can be prevented by minimizing shifts in blood volume. This indicates that any other effects of posture are of little importance as far as cardiac output and stroke volume are concerned. These findings extend the observations of Asmussen, Christensen and Nielsen (12), who found that the changes in the diuretic response to a water load, blood pressure, and pulse rate resulting from tilting could be prevented by inflating pneumatic cuffs around the thighs before tilting. Our observations are similar to those of Warren,

[1] The term "capillaries" is used here to refer to blood vessels into which $N_2O$ passes from the alveoli, although these may not meet the anatomical criteria for capillaries.

TABLE II

*A comparison between the reproducibility of duplicate determinations of cardiac output and stroke volume obtained by direct Fick and by the nitrous oxide technique* *

| | Direct Fick [Holmgren and Pernow (14)] | | | | Nitrous oxide (Present study) | | | |
|---|---|---|---|---|---|---|---|---|
| | n | $\bar{X}$ | ±SD | ±SD (% $\bar{X}$) | n | $\bar{X}$ | ±SD | ±SD (% $\bar{X}$) |
| Cardiac output, *L/min* | 17 | 7.7 | 0.89 | 11.6 | 30 | 4.6 | 0.34 | 7.4 |
| Stroke volume, *ml* | 17 | 84 | 10.2 | 12.1 | 30 | 68 | 5.0 | 7.3 |

* n = Number of pairs; $\bar{X}$ = mean; SD = standard deviation; SD (% $\bar{X}$) = standard deviation expressed as a percentage of the mean.

Weissler and Leonard, who studied the effects of changes in "central blood volume" on the cardiac response to atropine (8).

Because of the low pressure of the pulmonary circulation, the hydrostatic effects of posture have special hemodynamic significance. This has been clearly demonstrated by the use of radioactive $CO_2$ (9). Such studies have indicated that in the upright posture there are large differences in blood flow to different parts of the lung, the apexes receiving less than other parts. Such regional differences disappear in the horizontal posture. Reported studies of instantaneous pulmonary capillary blood flow indicate that the pattern of flow is pulsatile (10, 11). These studies, however, were performed when the subjects were sitting. One might postulate, therefore, that the pulsation of flow observed under these circumstances is a result of intermittent flow through the upper regions of the lung that occurs only at the time of peak driving pressure, whereas the flow through the capillaries at the bases of the lungs is constant throughout the cardiac cycle. Our observations indicate clearly that flow is pulsatile in both the upright and horizontal posture and that the pulsations thus cannot be due, to any large extent, to the hydrostatic effect of the upright posture. Posture, however, does influence the amplitude of the pulsations. This effect appears to be related to the coincident changes in stroke volume and not to the posture alone since, when stroke volume is kept constant by minimizing shifts in blood volume, posture has little effect on the pulsations.

## SUMMARY AND CONCLUSIONS

The effects of posture and distribution of blood volume on cardiac output, stroke volume, and instantaneous pulmonary capillary blood flow were studied in normal male subjects by a modification of the $N_2O$ technique. The results indicate that the changes in cardiac output and stroke volume that ordinarily accompany changes in posture can be prevented if shifts in blood volume are prevented. The results support the contention that the thoracic blood volume is an important determinant of the stroke output of the heart in normal resting man. Blood flow through the pulmonary capillaries is pulsatile in both the upright and horizontal postures. The amplitude of the pulsations is increased in the horizontal posture. This increase is related to coincident changes in stroke volume and not to the posture itself.

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