MECHANICAL EFFECTS OF THE HEART BEAT ON THE PRESSURE IN THE BODY PLETHYSMOGRAPH

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During suspended breathing, the pressure in the whole body plethysmograph undergoes rhythmic variations in time with the beat of the heart (Blair and Wedd, 1946; Lee and DuBois, 1955; Rigatto and Fishman, 1960). The major disturbance of the plethysmographic recording has been a transient increase in pressure at the time of ventricular systole. At the same point in the cardiac cycle there is also an indrawing of the thoracic cage (Blair and Wedd, 1939) and a movement of air into the lungs from the mouth (Bartlett, Brubach, and Specht, 1959).

These phenomena appear to be caused by a transient decrease in the intrathoracic blood volume during ventricular systole when the rate of aortic outflow exceeds that of the venous return, a conclusion reached by Blair and Wedd (1939, 1946) who have reviewed the early published reports. The effect of the sudden diminution in the intrathoracic blood volume is to lower the intrathoracic pressure. This in turn draws air inwards from the mouth, contracts the wall of the chest, and, in the presence of a finite airways resistance, temporarily expands the volume of air in the alveoli. It is this last effect that gives rise to the positive pressure wave in the plethysmograph.

If the above explanation were correct, it could be predicted that aortic incompetence and tricuspid incompetence would give rise to abnormally large pulsations in the plethysmograph, since both these conditions are associated with an excessive outflow of blood from the chest during systole and a correspondingly excessive inflow later in the cycle. The purpose of the present study was to examine the effects of these and other cardiac abnormalities on the plethysmographic record.

METHODS

All recordings were made on a four-channel Sanborn Poly-Viso recorder at a paper speed of 25 mm. per second. An electrocardiogram was recorded simultaneously from either lead I or II.

The body plethysmograph and its instrumental assembly have been described elsewhere (Mills, 1963). It is a modification of that published by Comroe, Botelho, and DuBois (1959). The pulsations that occurred in the plethysmograph were recorded at the maximal sensitivity available. To minimize the thermal drift of the recording at such a high amplification, all measurements were made with a 5 mm. bore opening between the plethysmograph and the atmosphere. A sine-wave pump of small stroke volume was used to calibrate the instrument, and the amplitude of the waves on the plethysmographic tracings was expressed in terms of the change in thoracic gas volume which would have caused them.

During most of the investigations, the subject breathed through a pneumotachograph (Lilly, 1950) which has a linear output to 350 l./min., but the recording of which was not calibrated. As well as providing a record of the movement of air at the mouth while ventilatory movements were suspended, this helped the investigator to determine the instant at which apnoea began and whether the glottis was closed or not.

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A shutter, which could be operated by means of a solenoid, was incorporated in the pneumotachograph assembly between the mouth and the flowmeter itself. When the shutter was closed, the pressure at the mouth could be recorded through a small side tube. Changes in pressure measured at the mouth were expressed in cm. water.

Movements of the wall of the chest were demonstrated by means of a mercury-in-rubber strain gauge. The gauge was part of a Wheatstone bridge, the output of which was amplified before it was applied to the recorder. The pneumograph was placed around the upper part of the chest to avoid interference from the apex beat. The same gauge was also used in two studies to detect jugular venous pulsations in patients with tricuspid incompetence. No attempt was made to calibrate this instrument.

RESULTS

Owing to the prolonged technical procedure, no attempt was made to carry out a complete study of every measurable effect in any one subject. In several of the subjects the glottis was involuntarily closed when ventilation was suspended, and so recordings with a patent airway could not be obtained. Other subjects had to be rejected because they were unable to hold their breath sufficiently steadily or for a sufficient length of time to establish consistent tracings over a number of cardiac cycles.

The nature of the tracing from the plethysmograph depended on the degree of inflation of the lungs as well as on the presence of certain cardiac abnormalities. The tracings recorded at the functional residual capacity will, therefore, be described and compared first, and the effects of varying the lung volume will be discussed later.

Normal Subjects at Functional Residual Capacity. Studies were performed on 8 normal people. Successful tracings were obtained with a patent airway on 5 of them. During early ventricular systole there was an increase in the plethysmographic pressure indicating an expansion of the volume of intrathoracic gas (Fig. 1). Timed from the peak of the R wave of the electrocardiogram, the

FIG. 1.—Normal subject with open glottis at three lung volumes: *left*, 2.3 l.; *centre*, 3.2 l.; *right* 5.7 l. Tracings from above downwards are: (1) pneumogram; upward deflection indicates decrease in girth; (2) plethysmograph pressure; 32 small squares equivalent to 7.5 ml.; (3) electrocardiogram.

increase in plethysmographic pressure started at an average of 0.07 sec. It had reached a maximum at 0.17 sec. and returned to the baseline at 0.30 sec. The mean duration of the wave was 34 per cent of the cardiac cycle which averaged 0.67 sec. in this group of subjects.

The amplitudes of the plethysmographic waves are given in the Table. In this group of normal subjects with an open glottis, the average amplitude was equivalent to a change of 0.94 ml. in the volume of gas in the chest.

When the glottis was open, the pneumotachograph tracing showed a sudden movement of air into the chest early in systole, at the time when the plethysmographic pressure was rising (see also Fig. 3 and 8).

TABLE

MAGNITUDE AND TIMING OF PULSATIONS IN THE PLETHYSMOGRAPH

Subjects	Closed airway					Open airway				
	T ₁ (sec.)	T ₂ (sec.)	T ₃ (sec.)	T ₄ (sec.)	Pulse amplitude (ml.)	T ₁ (sec.)	T ₂ (sec.)	T ₃ (sec.)	T ₄ (sec.)	Pulse amplitude (ml.)
<i>Normal</i>										
1	0.57	0.08	0.12	0.28	1.24	0.56	0.08	0.13	0.18	0.97
2	0.69	0.09	0.15	0.33	0.96	0.75	0.08	0.21	0.29	0.80
3	0.62	0.07	0.22	0.45	1.45	0.63	0.08	0.20	0.33	0.91
4	0.54	0.06	0.15	0.36	1.40	0.55	0.05	0.17	0.35	0.57
5	0.90	0.07	0.13	0.32	1.53	0.87	0.09	0.14	0.32	1.47
6	0.61	0.05	0.12	0.20	1.40					
7	0.85	0.03	0.15	0.40	1.28					
8	0.71	0.06	0.21	0.38	1.76					
<i>Aortic incompetence</i>										
1	0.52	0.06	0.17	*	5.87					
2	0.52	0.09	0.30	*	4.86					
3, before operation	0.66	—	0.21	*	6.67					
3, after operation	0.58	—	0.09	—	1.26					
4	0.74	0.04	0.26	*	8.73	0.72	—	0.26	0.42	5.44
<i>Tricuspid incompetence</i>										
1	0.82	—	0.34	—	4.83					
2	0.64	—	0.31	—	3.54					
3	0.78	—	0.33	—	9.95					
4	0.57	—	0.22	—	5.17					
5	0.62	—	0.32	—	4.15					
6	1.00	—	0.24	—	7.18					
<i>Pulmonary stenosis</i>	0.61	0.02	0.10	0.24	1.53	0.61	0.02	0.09	0.21	1.04
<i>Aortic stenosis</i>	1.05	0.03	0.10	0.20	0.89					
<i>Atrio-ventricular dissociation</i>	2.06	0.05	0.10	0.50	4.24					
<i>Mitral stenosis</i>	0.58	0.04	0.15	0.32	1.30					
<i>Mitral stenosis</i>	0.62	0.09	0.24	0.30	1.23					
<i>Mitral stenosis and incompetence</i>	—	—	—	—	—	0.53	0.12	0.19	0.30	0.96

T₁=duration of cardiac cycle.T₂=R wave to commencement of rise in plethysmograph pulse.T₃=R wave to peak of plethysmograph pulse.T₄=R wave to completion of plethysmograph pulse.

* =beginning of next pulse.

FIG. 2.—Normal subject at three levels of inspiration with an occluded external airway. Tracings from above downwards are: (1) plethysmograph pressure; calibration shown; (2) electrocardiogram.

Recordings were made on 8 normal people with a closed glottis. The details are given in the Table, and illustrated in Fig. 2 and 16. In the 5 subjects from whom recordings had also been obtained with an open glottis, the amplitude of the systolic pulse wave was consistently greater with a closed glottis (see also Fig. 3 and 8). The average change in the volume of gas in the chest was 1.32 ml. with a closed glottis, compared with 0.94 ml. with an open glottis. There was no measurable

FIG. 3.—Aortic incompetence at functional residual capacity. *Left*=open airway; *right*=closed airway. Tracings from above downwards: (1) plethysmograph pressure; 29 small squares equivalent to 7.5 ml.; (2) left, pneumotachograph, inspiration downward; right, mouth pressure; 15 small squares=2 cm. water; (3) electrocardiogram.

difference in the time of onset of the plethysmographic pulse wave. In 4 out of the 5 subjects, the peak of the wave came slightly earlier when the glottis was closed, the mean time from the peak of the R wave being 0.15 sec. In 4 out of these 5 subjects the duration of the wave was greater when the glottis was closed, the mean time between the R wave and the end of the plethysmographic wave being 0.35 sec.

Variations in the intrathoracic pressure, reflected by the pressure at the mouth with a closed shutter, were recorded in 4 normal people. The major deflection was a transient fall in pressure occurring during systole and at the same time as the increase in plethysmographic pressure (see also Fig. 3, 6, 8, and 15). The maximal fall in pressure at the mouth in each of these four subjects was 0.16, 0.19, 0.28, and 0.33 cm. water.

The oesophageal pressure was recorded in 2 subjects but the readings were dominated by the mechanical effects of the movement of the heart.

Movements of the chest wall were recorded by the pneumogram in 2 subjects. The tracings showed that there was a sudden decrease in girth during systole (Fig. 1) at the time when the plethysmographic pressure was increasing. The subsequent outward movement of the chest occurred more slowly.

Aortic Incompetence at Functional Residual Capacity. Four patients with a serious degree of aortic incompetence and minimal aortic stenosis were studied. In 3 of these the diagnosis was confirmed at operation. In only one patient were results obtained with an open airway (Fig. 3). Satisfactory recordings were made on all 4 with a closed airway and the plethysmograph showed a prominent systolic wave of increased pressure. The wave started at an average time of 0.05 sec. after the peak of the R wave of the electrocardiogram. The upstroke of the wave was steep and the peak was reached at an average time of 0.24 sec. after the R wave, which was longer than that observed in normal subjects. For the rest of the cardiac cycle the pressure in the plethysmograph gradually declined. The average amplitude of the pulse wave was equivalent to a change of 6.53 ml. in the volume of gas in the chest. The individual values are given in the Table and in Fig. 16.

Figure 3 shows how the pulse wave in the plethysmograph increased in amplitude when the external airway was obstructed by the shutter. The shape of the wave also became more angular with a closed glottis. The pneumotachograph record in the first half of Fig. 3 shows that the sys-

tolic wave in the plethysmograph is accompanied by a sudden movement of air into the chest. There appears to be a similar small movement of air associated with atrial systole, but no accompanying wave appears on the plethysmogram. The second half of Fig. 3 shows that, when the shutter of the pneumotachograph was closed, the pressure in the plethysmograph increased. The fall in pressure at the mouth during systole was 2.33 ml. water, which is considerably more than that found in the normal subjects. Figure 4 shows that the movements of the chest wall in aortic incompetence were similar to those seen in normal people. In this recording, the pressure in the plethysmograph during early diastole swung below the presystolic level.

In one patient, measurements were made before and after the aortic valve had been repaired (Fig. 5). Before the operation the amplitude of the wave in the plethysmograph was equivalent to an increase of 6.67 ml. in the volume of gas in the chest. After the operation this had diminished to 1.26 ml., which is within the normal range.

Tricuspid Incompetence at Functional Residual Capacity. Recordings were carried out on 6 patients in whom a clinical diagnosis of tricuspid incompetence had been made. The airway was closed in every case. As in the patients with aortic incompetence, the plethysmogram showed a pulse wave of larger amplitude than normal, equivalent on the average to a change of 5.80 ml. in the gas in the chest (Fig. 16). The shape of the systolic wave (Fig. 6) was in general rounder than that seen in aortic incompetence. In most instances there was evidence of a diastolic dip in the plethysmogram before the main systolic rise. For this reason it was difficult to ascertain the exact points at which the systolic wave in the plethysmogram began and ended. The peak of the systolic wave in the plethysmogram could be identified more accurately and occurred at a mean time of 0.29 sec. from the peak of the R wave on the electrocardiogram. This was longer than the average value for the patient with aortic incompetence and considerably longer than that found in normal subjects.

FIG. 4.—Aortic incompetence at functional residual capacity. Tracings from above downwards are: (1) pneumogram; downward deflection indicates decrease in girth; (2) plethysmograph pressure; calibration shown; (3) electrocardiogram.

FIG. 5.—Aortic incompetence at functional residual capacity. *Left*, before operation; *right*, after operation. Tracings from above downwards are: (1) plethysmograph pressure; 31 squares equivalent to 7.5 ml. water; (2) electrocardiogram.

FIG. 6.—Tricuspid incompetence with occluded airway at three levels of inspiration. Tracings from above downwards are: (1) plethysmograph pressure; calibration shown; (2) mouth pressure; calibration shown; (3) electrocardiogram.

The pressure at the mouth with a closed shutter was recorded in 2 patients (Fig. 6). The positive systolic wave in the plethysmograph was accompanied by a fall in the pressure at the mouth of 2.73 and 1.82 cm. water. These figures are considerably larger than those found in normal subjects.

In 2 patients the pulsations in the neck were recorded by means of the mercury-in-rubber strain gauge. Figure 7 shows that, at the functional residual capacity, the systolic expansion in the neck was synchronous with the systolic pressure wave in the body plethysmograph.

Other Abnormalities at Functional Residual Capacity. (i) *Pulmonary valve disease.* One patient with an isolated pulmonary valvular stenosis was studied. At cardiac catheterization there was a difference of 95 mm. Hg between the systolic pressure in the right ventricle and that in the pulmonary artery.

FIG. 7.—Tricuspid incompetence with closed glottis at three lung volumes: *left*, low; *centre*, functional residual capacity; *right*, high. Tracings from above downwards are: (1) plethysmograph pressure; 30 small squares equivalent to 7.5 ml.; (2) girth of neck; upward deflection indicates expansion; (3) electrocardiogram.

FIG. 8.—Pulmonary stenosis at functional residual capacity. *Left*, closed airway; *right* open airway. Tracings from above downwards are: (1) plethysmograph pressure; 31 small squares equivalent to 7.5 ml.; (2) *left*, mouth pressure; 19 small squares is 2 cm. water; *right*, pneumotachograph; inspiration downward.

Tracings were obtained with a closed and an open airway. The pulsations seen in the plethysmogram were comparable to those found in normal subjects. The inward flow of air concurrent with the rise in the plethysmogram is shown in Fig. 8. The increase in the magnitude of the plethysmographic pulsations when the airway was occluded and the fall in pressure at the mouth during systole are shown on the same figure.

Studies were made on one patient with an isolated pulmonary valvular incompetence. The recordings were not technically satisfactory, but it was apparent that the deflections on the plethysmogram were not increased.

(ii) *Aortic stenosis*. One subject with aortic stenosis was studied. At left ventricular puncture the systolic pressure in the left ventricle exceeded that in the brachial artery by 100 mm. Hg. The appearance of the plethysmogram was similar to that of the normal group but was small in amplitude. The tracing, which is shown in Fig. 9, shows a presystolic wave, possibly related to atrial systole.

(iii) *Mitral valve disease*. Three patients were studied: two with mitral stenosis and one with both stenosis and incompetence. The detailed measurements are given in the Table and the tracings from the patient with stenosis and incompetence are shown in Fig. 10. The systolic wave on the plethysmogram was within normal limits in each case.

(iv) *Atrio-ventricular dissociation*. One patient with atrio-ventricular dissociation was studied. The ventricular rate was 29 a minute. The tracing from this patient is shown in Fig. 11. The amplitude of the systolic pulse was equivalent to an increase of 4.24 ml. in the volume of gas in the chest, which is abnormally large. The pulse wave was also sustained for a longer time than normal. During the early part of the prolonged diastolic phase, the pressure in the plethysmograph fell to its lowest point which was succeeded by a gradual increase as diastole continued. There were no pulsations synchronous with the atrial beat.

FIG. 9.—Aortic stenosis with closed glottis at three lung volumes: *left*, low; *centre*, functional residual capacity; *right*, high. Tracings from above downwards are: (1) plethysmograph pressure; 28 small squares equivalent to 7.5 ml.; (2) electrocardiogram.

FIG. 10.—Mitral stenosis with open glottis at three lung volumes: *left*, low; *centre*, functional residual capacity; *right*, high. Tracings from above downwards are: (1) plethysmograph pressure; 30 small squares equivalent to 7.5 ml.; (2) pneumotachograph; inspiration downwards; (3) electrocardiogram.

FIG. 11.—Atrio-ventricular dissociation with closed glottis at functional residual capacity. Tracings from above downwards are: (1) plethysmograph pressure; 30 small squares equivalent to 7.5 ml.; (2) mouth pressure; 16 small squares, 2 cm. water; (3) electrocardiogram.

FIG. 12.—Constrictive pericarditis with closed glottis at functional residual capacity. Tracings from above downwards are: (1) plethysmograph pressure; 30 small squares equivalent to 7.5 ml.; (2) electrocardiogram.

FIG. 13.—Constrictive pericarditis with closed glottis at three lung volumes: *left*, low; *centre*, functional residual capacity; *right*, high. Tracings from above downwards are: (1) plethysmograph pressure; 29 squares equivalent to 7.5 ml.; (2) mouth pressure (lack of deflections indicates a closed glottis); (3) electrocardiogram.

(v) *Constrictive pericarditis*. Two patients were studied, in both of whom the diagnosis was confirmed at operation and ultimately at necropsy. In one patient (Fig. 12) the plethysmogram showed very large deflections similar to those seen in tricuspid incompetence. The appearance was due to a combination of an abnormally large positive systolic wave and the presence of a deeply negative component during diastole. The recording from the other patient was within the normal limits at the functional residual capacity, but developed an abnormally deep negative wave in diastole at a low lung volume (Fig. 13).

The Effects of Inspiratory Position. Although, in the majority of subjects, the plethysmogram at functional residual capacity showed only one positive wave during the cardiac cycle, there were instances in which two positive waves were observed. The second wave started at about the end of the T wave on the electrocardiogram. Further investigation of this wave showed that its appearance depended on the degree of inflation of the lungs. The inspiratory position of the chest often also had an influence on the size of the first, systolic, positive wave on the plethysmogram.

At a lung volume approaching the residual volume, the systolic positive wave on the plethysmogram, which has been considered up to this point, was usually taller and broader than at the functional residual capacity (Fig. 1, 2, 9, and 10) and sometimes slightly bifid. At the same time the diastolic phase of the plethysmogram often became slightly negative relative to the pressure recorded at the beginning of systole (Fig. 1, 9, and 14). In the patient with constrictive pericarditis, the diastolic fall was particularly large (Fig. 13).

At the functional residual capacity, the systolic positive wave on the plethysmogram was as has been described up to this point, and during diastole the recording was substantially flat (Fig. 1, 2, 9, 10, 13, and 14).

At a lung volume approaching the inspiratory capacity, the systolic positive wave on the plethysmogram usually, but not always, diminished in size (Fig. 1, 2, 9, and 13) and occasionally could not be distinguished. The second positive wave on the plethysmogram now usually became apparent (Fig. 1, 2, 9, 10, 13, and 14). It started towards the end of the T wave on the electrocardiogram and lasted for a variable proportion of the cardiac cycle, sometimes continuing to the end of diastole. The magnitude of the second wave frequently exceeded that of the first wave.

FIG. 14.—Normal subject with closed glottis. Four lung volumes from left to right: low, functional residual capacity, high, and maximal inspiration. Tracings from above downwards are: (1) plethysmograph pressure; 30 small squares equivalent to 7.5 ml.; (2) electrocardiogram.

FIG. 15.—Normal subject with closed external airway at high lung volume. Tracings from above downwards are: (1) plethysmograph pressure; 29 small squares equivalent to 7.5 ml.; (2) mouth pressure; 15 small squares, 2 cm. water; (3) electrocardiogram.

FIG. 16.—A comparison of the amplitude of the pulse wave in the body plethysmogram in normal subjects, patients with tricuspid incompetence, and patients with aortic incompetence. The interrupted line with an arrow shows the change that occurred after a repair of aortic incompetence.

the chest, which the evidence suggests are the cause of the variations in pressure, are given not by the absolute pressure in the plethysmograph but by its rate of change. And so, to give an immediate representation of the net rate of movement of intrathoracic blood, the plethysmographic tracing would have to be differentiated with respect to time. A positive wave on the plethysmogram, for instance, represents not only a net flow of blood out of the chest during the upstroke but also a net flow of blood into the chest during the downstroke; while the wave itself represents the decrease and subsequent increase in the volume of blood remaining in the chest.

The production of such a wave on the plethysmogram depends on the presence of a finite airways resistance, and it would be expected that the amplitude of the wave would increase when the glottis was closed or the airway obstructed. An increase in the volume of air in the lungs or a decrease in the compliance of the chest wall would also be expected to increase the amplitude of the pulse wave in the plethysmograph, while inertial and resistive forces within the tissue of the thorax may modify the wave. It follows from these considerations that, though it is possible to calculate the change in the volume of gas in the chest from the amplitude of the wave in the plethysmograph, it is not possible to calculate the underlying change in intrathoracic blood volume.

The second wave, like the first, was accompanied by a movement of air into the lungs (Fig. 10), and by a fall in the pressure at the mouth when the pneumotachograph shutter was closed (Fig. 15).

The second wave of the plethysmogram could also be made to appear during deep inspiration in patients with aortic incompetence. In patients with tricuspid incompetence, only one sustained positive wave could be discerned on the plethysmogram at any level of inspiration. However, during deep inspiration, the onset of this wave moved from shortly after the R wave to the region of the T wave on the electrocardiogram (Fig. 6 and 7). Hence the underlying mechanism seems to be the same as in the normal subjects. In the deep inspiratory position the delayed positive wave on the plethysmogram was still accompanied by a fall in pressure at the mouth when the pneumotachograph shutter was closed (Fig. 6). By contrast, the timing of the systolic venous wave recorded in the neck in these patients remained unchanged. Thus the pulse wave in the neck and that in the plethysmograph became asynchronous (Fig. 7). Direct recordings of the right atrial and brachial arterial pressure waves showed that these occurred at the same time after the R wave on the electrocardiogram, whatever the inspiratory position.

DISCUSSION

The changes in pressure in the plethysmograph correspond to the changes that occur in the volume of gas in the chest. Nevertheless, the net rates of flow of blood into and out of

The pulsatility of the exchange of oxygen and carbon dioxide in the alveoli such as has been demonstrated by Bosman, Lee, and Marshall (1965), could also influence the recording from the plethysmograph. We have had the privilege of sharing our observations with Dr. Lee and Dr. Bosman who have been carrying out very similar investigations in Oxford. Their studies (Bosman and Lee, 1965) have shown that the pulsatile exchange of gases in the alveoli does not substantially alter the shape of the plethysmographic waves caused by the mechanical action of the heart.

The increase in the plethysmographic pressure at the beginning of systole is accompanied by a decrease in the intrathoracic pressure, a movement of air into the chest from the mouth, and a decrease in the girth of the chest. This combination of events supports the contention that at the beginning of systole there is a sudden decrease in the volume of blood in the chest. Further support comes from the distinctive increase in the positive systolic plethysmographic wave which occurred in patients with aortic incompetence and tricuspid incompetence. A similar increase in this wave occurred in the patient with atrio-ventricular dissociation, a condition that is associated with a high stroke volume due to the slow beat. The wave in the patient with aortic stenosis was of lower amplitude than normal, a finding which is consistent with a diminished rate of aortic flow in early systole.

Neither hypertrophy of the right ventricle due to pulmonary stenosis nor hypertrophy of the left ventricle due to aortic stenosis caused any increase in the systolic wave on the plethysmogram. The findings in the patients with mitral valve disease were also normal. This was so even in the patient with mitral incompetence. It could be explained on the grounds that in mitral incompetence the regurgitation simply causes a movement of blood within the thorax, while in tricuspid incompetence the regurgitating blood leaves the thorax and thus decreases the volume of blood there. Similar considerations apply to pulmonary valvular incompetence.

The movement of blood into and out of the chest, which is responsible for the changes in the volume of blood there, follows a complex pattern the nature of which is only partly known. The shape of the flow curve in the aorta has been reviewed by McDonald (1960). The shape of the flow curve in the venæ cavæ has been reviewed by Brecher (1956) and investigated in man by Müller and Shillingford (1955). During ventricular systole there is sudden increase in the flow of blood along the aorta which would cause a diminution in the volume of blood in the chest. On the other hand, ventricular systole, by pulling down the atrio-ventricular ring, also causes a substantial increase in the flow of blood through the venæ cavæ into the chest. Such a movement would cause an increase in the volume of blood in the chest. Hence, the arterial and venous effects of ventricular systole are, in this respect, opposed to each other. Presumably, at the beginning of ventricular systole, the rate of aortic outflow exceeds that of caval inflow, while towards the end of systole the situation has become reversed.

During diastole, the rates of flow of blood into and out of the chest are lower than during systole, and, judging by the relatively flat appearance of the plethysmogram, they seem largely to cancel each other out at the functional residual capacity. Occasionally, however, a small positive pre-systolic wave could be distinguished, which might be due to a sudden diminution in venous return at the time of contraction of the right atrium.

The changes in the plethysmogram that occur at different levels of inspiration are more difficult to explain. Inspiration is associated with an augmented venous return and the successive diminution in the positive systolic plethysmographic wave with an increasing degree of inspiration may be due to an increasing systolic venous inflow. On the other hand, in the presence of tricuspid incompetence, deep inspiration could abolish the systolic rise in plethysmographic pressure while the systolic venous wave in the neck was unaffected.

This dissociation between the hæmodynamic and plethysmographic effects of inspiration suggests the possibility that some other mechanical factor is operating. The presence of a large volume of air in the lungs would, by itself, be expected to enhance the pulse-waves in the plethysmograph and not diminish them. On the other hand, the effect of the elastic recoil of the chest wall is such that a

compensatory decrease in girth with systole is more likely to occur at a high lung volume, provided that the sustained contraction of the inspiratory muscles will allow it to do so.

There are three possible mechanical effects of the heart beat which could also influence the volume of gas in the chest at different levels of inspiration in a manner consistent with the observed alterations in the plethysmogram. Studies on the distensibility of rabbits' lungs have shown that raising the pulmonary arterial pressure tends to distend the alveoli at a low lung volume and to compress them at a high lung volume (Harris, 1955). It is possible that the systolic increase in pulmonary arterial pressure could have this effect. Another conceivable mechanical effect of ventricular systole is an uncoiling of the aorta which would tend to increase the antero-posterior diameter of the chest particularly at a low lung volume. Thirdly, it is possible that, during ventricular contraction, the heart actively pulls the diaphragm upwards and that this action is greatest in the position of deep inspiration when the diaphragm has descended.

All such mechanical effects of ventricular systole would favour the systolic expansion of the intra-thoracic gas in the expiratory rather than the inspiratory position. In this way the positive systolic wave on the plethysmogram would be augmented in expiration and diminished in expiration.

The same mechanisms could also conceivably explain the second positive plethysmographic wave which develops on deep inspiration. The upstroke of this wave represents an expansion of the gas in the chest and is accompanied by a fall in the intrathoracic pressure and a movement of air into the lungs from the mouth. It is difficult to explain this on hæmodynamic grounds since it occurs at a time when the rate of aortic outflow and venous inflow are low. According to the recording of Müller and Shillingford (1955) there may be some slight reversal of the caval inflow at this time; but equally, according to the view of McDonald (1960), there may be some reversal of the aortic flow.

Should the gas in the lungs be squeezed during systole by the pulmonary arterial tree tending to take up its natural position or by the diaphragm being pulled upwards, then it is feasible that the reverse will occur after the end of systole. In this way there could be a sudden expansion of the alveolar gas at the beginning of diastole. Subsequently the wave of increased venous return that follows the opening of the tricuspid valve would compress the alveolar gas and cause the plethysmographic pressure to decrease again.

SUMMARY

Recordings of pressure in the body plethysmograph show a positive wave synchronous with ventricular systole in normal people at the functional residual capacity of the lungs. The wave is accompanied by a diminution in intrathoracic pressure, a flow of air into the lungs from the mouth and a decrease in the girth of the chest. The amplitude of the wave is increased in patients with aortic incompetence and tricuspid incompetence. It is not affected by disease of the mitral or pulmonary valves.

The wave is an expression of an expansion of the volume of gas in the lungs which would seem to be caused by a transient decrease in the intrathoracic blood volume during ventricular systole.

The shape of the plethysmographic tracing is modified in a complex fashion by changes in the inspiratory position.

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