Postural Effects on the Baselines of Ventricular Performance

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In both dogs and human subjects, fully relaxed in the horizontal position, the ventricular dimensions and stroke volume are generally at or near maximal levels. During exertion the stroke volume does not progressively increase, indeed it rarely exceeds recumbent control values. On sitting or standing the ventricular size and stroke volume diminish to a new lower control level. When control values are measured during quiet standing, exertion produces a slight but consistent increase in stroke volume approximately to the level found in recumbency. This difference in the baseline for control values must be considered in evaluating the cardiovascular response to exercise.

Most cardiovascular investigation is conducted on animals and human beings relaxed in recumbent positions to provide stable baseline conditions with long columns of blood oriented horizontally and the stresses on the cardiovascular system presumably at a low, constant level. Changes in performance of the ventricular chambers have been studied in intact dogs while they were running down a hall or on a motor-driven treadmill at 3 m.p.h. on a 5 per cent grade. During the first few series of experiments, a stable control baseline was achieved by allowing the dogs to remain reclining until just before the exercise began. Under these conditions the left ventricular dimensions were observed to diminish during the exertion. Subsequently, continuous recordings of left ventricular diameter, circumference, and length have all demonstrated that if the dogs are standing at the onset of exercise, the ventricular diastolic dimensions may diminish slightly, increase slightly, or most commonly, remain unchanged. The systolic deflections were usually slightly increased by more complete systolic ejection.

A survey of recent data on human subjects revealed that an increase in stroke volume was neither an essential nor a consistent feature of the cardiac response to exertion. This conclusion was in conflict with the observation by Mitchell, Sproule, and Chapman that stroke volume consistently increases about 2-fold from erect control values to maximal exertion. We are indebted to Chapman for his suggestion that this apparent discrepancy might reflect differences in control values, measured in the recumbent position in some studies and in the erect position in others.

Thus, conflicting conclusions have resulted from failure to recognize the nature of the change in baselines of cardiac performance that occur during a change from erect to recumbent position. In other words, it is important to appreciate the differences between the baselines of cardiac performance in erect and reclining human subjects and dogs.

Methods

In the experiments on intact unanesthetized dogs, left ventricular performance was continuously analyzed in terms of several parameters during spontaneous activity. The technics employed for these studies have been described elsewhere and are only summarized briefly here. Changing left ventricular dimensions were recorded continuously by means of gages applied directly to the ventricle during aseptic surgery. A modified sonar technic was employed to record left ventricular diameter. Wires were led through the back, so that recordings could be made at will after recovery from the operation.

Effective left ventricular pressure was recorded through an indwelling cannula extending from the
left atrium through the posterior thoracic wall. A polythene catheter (about 13 cm. long) from a miniature differential transformer pressure transducer was threaded through this cannula into the left ventricular cavity. From a balloon near the heart, intrapleural pressure was impressed upon the back of the gage, which then responded to effective ventricular pressure. Left ventricular pressure, amplified 4 times, was recorded to demonstrate changes in diastolic pressure.

Heart rate was indicated by a ratemeter triggered by the rapid rise of the ventricular pressure. During each cycle the galvanometer deflection indicated the reciprocal of the cycle length of the preceding cardiac cycle.

**RESULTS**

In dogs relaxed in the prone position, the heart rate tended to be slow (70 to 100 per minute) and sinus arrhythmia was frequently apparent (fig. 1A). The left ventricular diameter increased very rapidly during early diastole and reached a plateau. However, the diameter often continued to increase very gradually, even when the diastolic interval was very long. Atrial contraction contributed very little to ventricular filling under these conditions because the dimensions approached maximal distention during the rapid filling phase. When the dog stood quietly with his trunk oriented horizontally, the diastolic and systolic dimensions were both promptly reduced and the amplitude of the systolic deflection was decidedly diminished (fig. 1B). The reduction in diastolic distention was accompanied by a reduction in diastolic filling pressure of no more than 2 mm. Hg. The hydrostatic column from the ventricle to the gage was exactly the same in the 2 records. In many instances no change in effective filling pressure was demonstrable.

The longest cardiac cycle in the standing record was longer than the shortest cycle in the reclining record, so the difference in diastolic and systolic dimensions in the 2 positions was not due solely to filling time. The ventricle filled much more rapidly in the standing position than in the reclining position. True diastasis was rarely noted in the standing position except during the compensatory pause following premature contractions. The relative degree of distention during early diastole was much smaller in the standing position. All these factors suggested that the distensibility of the ventricle was significantly different in the standing and reclining dog even though the trunk was horizontally oriented in both cases.

During the transition from the standing (or sitting) position to the reclining position, the diastolic and systolic diameter increased progressively with each beat until a plateau was attained after 5 or 6 cycles. As the end-diastolic diameter became larger and larger, the amplitude of the systolic excursion increased correspondingly.5 This observation suggested that stroke volume and stroke work increased with greater diastolic distention, as would be predicted by Starling’s law of the heart. Such a relationship between diastolic distention and systolic excursions was consistently observed during the transition from sitting or standing to the recumbent position.5

Reduction in diastolic and systolic left ventricular dimensions was produced consistently by passive tilting of 5 dogs by gently elevating the head end of a tilt board to approximately 30 degrees. Even when the animals did not stir, the left ventricular dimensions promptly diminished to a lower level, which was well maintained until the animal was returned to the horizontal position (fig. 2A). Tilting the dog with the head...
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down 30 degrees, produced a similar response although this could easily be obscured if the animal was not fully relaxed during the control period (fig. 2B). The orientation of the body itself was not necessarily the prime factor in this reaction. If the dog was relaxed in the prone position and merely lifted his head in response to noise or movement elsewhere in the room, the ventricular dimensions usually diminished promptly.

Passive tilting of human subjects is known to produce a diminution in cardiac output and in stroke volume. As an example, a reduction in stroke volume associated with an increase in heart rate after tilting normal subjects head up to 60 degrees was replotted from the study of Weissler et al. (fig. 2C). The reduction in stroke volume was probably greater during a passive tilt than during quiet standing because of the contraction of the weight-bearing muscles.

The difference between recumbent and standing baselines assumed importance in evaluating the left ventricular response to exertion (fig. 3). If a recumbent dog stood up just before he began to exercise on a motor-driven treadmill (fig. 3B), the left ventricular diameter abruptly diminished and effective filling pressure diminished at the same time. Under these conditions, the left ventricular response to exertion involved marked reduction in diastolic and systolic diameter along with an obvious reduction in systolic deflection with each cycle. The heart rate increased from about 90 to about 200 per minute. A few minutes later, the exercise was repeated, but this time the dog was standing during the control period (fig. 3D). The diastolic diameter was essentially unchanged and the stroke deflection was slightly increased by greater systolic ejection during exertion.

Although the increase in stroke was slight and the acceleration of the heart rate was great during exercise, the ventricular response must not be visualized as merely an increase in heart rate. The importance of this injunction was clearly demonstrated by artificial induction of tachycardia in the same animal while it was reclining quietly on a table. A pair of electrodes had previously been sutured to the atrial wall with wires leading to the outside. The changes in heart rate during a previous exercise response had been recorded and stored with a magnetic tape recorder. When this tape was replayed through a stimulator connected to the pacemaker electrodes on the atrium, the changes in heart rate that had occurred during a previous exercise could be reproduced precisely (fig. 3E). Under these conditions, the diastolic diameter was profoundly reduced and the stroke deflections were also greatly reduced. Incidentally, the ventricular systolic pressure was also diminished in contrast with the elevation in pressure generally observed during exercise. Thus, although the increased cardiac output during exertion is dependent primarily on an acceleration of the heart rate, this adjustment apparently must be accompanied by adaptations in the peripheral circulatory beds and in functional characteristics of the myocardium, i.e., contractility.

In previous publications, evidence from 7 different series of experiments was presented to support the conclusion that an increase in stroke volume was not an essential feature of the ventricular response to exercise in normal human subjects. In some of these experiments, the subjects performed leg exercises while reclining on a fluoroscopic table, some performed exercises while seated on a bicycle ergometer, and others walked or ran on motor-driven treadmills. The potential importance of posture not having been recognized in these responses, individual values for stroke volume, heart rate, and arteriovenous oxygen difference were plotted against oxygen consumption without regard for the posture of the subject during control and exercise determinations. In retrospect, this practice appears to be unwise in view of the importance of posture in the experiments on dogs illustrated in figure 3.
Fig. 2 Top. A. Reduction in diastolic and systolic diameter and stroke deflection on passive tilting of relaxed conscious dogs with the head up 30 degrees. Top, left ventricular diameter; middle, left ventricular pressure; bottom, diastolic pressure. B. Passive tilting with the head down 30° also produced a reduction in ventricular dimensions and systolic excursions. However, in this case the animal was not well relaxed during the control period but was relaxed.

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To examine the influence of position on the changes in stroke volume, mean values from studies on recumbent and erect human subjects were plotted separately in figure 4A. Since oxygen consumption was recorded as milliliters per minute in some studies and as milliliters per minute per square meter in others, 2 scales are indicated on the abscissa. The studies by Donald et al.\(^7\) and Barratt-Boyes and Wood\(^8\) revealed no evidence of a progressive increase in mean values for stroke volume during exercise by recumbent subjects. On the other hand, Dexter et al.\(^9\) reported data indicating a slight increased in stroke volume that were lower during the control period prior to exertion. The single exception was the experiment of Asmussen and Nielsen,\(^11\) in which the control values were measured in the recumbent position and were not obviously different from values during levels of exertion increasing oxygen consumption as much as 10-fold. These data are consistent with the view that stroke volume remains essentially unchanged over a very wide range of physical exertion. Even at peak levels of exercise, the stroke volume did not consistently exceed recumbent control values.

The evidence that stroke volume did not increase progressively as the intensity of exertion was increased appeared inconsistent with the data reported by Mitchell, Sproule, and Chapman,\(^2\) who found that stroke volume exceeded control values by about 2-fold during intense exercise that produced a maximum oxygen consumption. In this study, Mitchell et al. measured oxygen consumption during progressively increasing work loads lasting 2½ minutes on a motor-driven treadmill running at 6 m.p.h. with the grade being elevated in increments of 2.5 per cent until oxygen uptake per minute leveled off. In some subjects, it was necessary to increase the speed as well as the grade and the maximum work load was 9 m.p.h. at 14.75 per cent grade. At a later date, both maximal oxygen consumption and cardiac output (by an indicator-dilution technic) were measured at rest and at 3 work loads. These data had not been included in the previous reports\(^1,6\) because it seemed inappropriate to plot heart rate or stroke volume against values of oxy-

following return to the horizontal position. C. In normal human subjects passively tilted 60° head up, stroke volume decreased and mean cardiac output diminished from 5.9 to 5.0 L. per minute in spite of accelerated heart rate (after Weissler et al.\(^13\)).

Fig. 3 Bottom. A. Left ventricular diameter approached maximal dimensions in a recumbent dog. B. The diastolic and systolic dimensions diminished promptly on standing and during exercise on a treadmill at 3 m.p.h. on a 5 per cent grade. D. Little change in ventricular diameter during exercise following a standing control period (C). E. Artificially induced tachycardia caused a pronounced diminution of diastolic diameter and reduced systolic excursion.
gen consumption that had been pushed to a plateau (i.e., oxygen debt was accumulating rapidly). The mean control value for stroke volume of subjects standing on the treadmill were lower than most control values and considerably below the recumbent control values reported by Asmussen and Nielsen. 

The 3 factors that contribute to increased oxygen delivery to the tissues during exercise include stroke volume, heart rate, and oxygen extraction from the blood (arteriovenous oxygen difference). The relative contributions of these 3 factors are illustrated in figure 5, in which data from 10 different series of experiments have been compiled and identified with reference to the posture of the subjects during control measurements and exercise. In addition to the data previously reported, this graph includes individual points from the study by Mitchell et al., plus previously unreported data from subsequent studies. Data from the studies by Riley et al. and Freedman et al. have also been included.

The stroke volume was surprisingly uniform over the wide range of exertion and oxygen consumption. In general, the values from erect subjects were lower at rest and slightly higher at maximal levels of exertion. The heart rate increased progressively to levels between 160 and 200 beats per minute and leveled off. The arteriovenous oxygen difference apparently increased progressively over the full range of oxygen consumption.

Our interpretation of the contribution of stroke volume, heart rate, and arteriovenous oxygen difference to the total oxygen delivery during physical exertion are indicated by

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**Fig. 5.** The contribution of (A) stroke volume, (B) heart rate, and (C) arteriovenous oxygen difference to the delivery of oxygen during exertion. **Dotted lines,** erect subjects; **Continuous lines,** recumbent subjects. In the key, the symbols represent the following references: a, 2; b, 12; c, 14; d, 13; e, 11; f, 7; g, 8; h, 9; i, 15; j, 10.
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lines, drawn by eye, through the points on each graph and superimposed on a single chart (fig. 5D). Among most subjects studied while recumbent, stroke volume changed very little, as indicated by the light continuous line. In general, control values for erect subjects, indicated by the interrupted lines, revealed a faster heart rate and smaller stroke volume than those of recumbent subjects. Stroke volume of subjects exercising in the erect position did not increase above values typically observed in recumbent subjects until maximal oxygen consumption was attained (interrupted lines). Thus, stroke volume during exercise may increase in erect subjects primarily because their control values were lower than those of recumbent subjects. There does not appear to be a progressive increase in stroke volume associated with greater oxygen delivery or levels of physical exertion.

DISCUSSION

Inattentive, recumbent dogs have heart rates generally ranging between 70 and 100 beats per minute and under these conditions, the left ventricle tends to function at or near its maximal dimensions as recorded by diameter, length, and circumference gages. In such circumstances, intravenous infusions of blood sufficient to elevate effective filling pressure by as much as 15 mm. Hg failed to increase left ventricular diameter by as much as 1 mm. Any change in the status of the animal, including the administration of anesthetics, causes a reduction in the ventricular dimensions. Thoracotomy tends to reduce further the size of the cardiac chambers. Thus, the exposed heart of an anesthetized dog functions at a baseline different from that for the recumbent intact animal. When an intact healthy dog stands up, his ventricular size diminishes, but this decrease does not necessarily imply that the new baseline corresponds to that for the anesthetized thoracotomized animal. Thus, at least 3 different baselines may require consideration in evaluating the physiology of cardiac function.

The change in size of the heart found in dogs has a direct counterpart in human subjects. For example, Sjöstrand reported roentgenographic studies demonstrating that size of the cardiac silhouette was maximal in the recumbent position. Any change in the status of the individual induced either a reduction in heart size or no change. He was unable to cause the heart to expand beyond those dimensions. The fact that stroke volume and cardiac output diminish as a result of passive tilting with the head up indicates that a reduction in stroke volume of smaller magnitude apparently occurs when average normal subjects stand up (figs. 3 and 4). During control determinations in erect subjects, the heart rate tended to be somewhat higher and the stroke volume appeared to be lower. In addition, anticipation of violent exercise may also be accompanied by a further cardioacceleration. The effect of this acceleration may correspond to the reduction in systolic deflections produced by experimentally induced tachycardia in dogs (fig. 3E). However, this is not the whole story because Barger et al. reported that changes in cardiac output produced in resting dogs by excitement were frequently as large as those induced by moderate exercise. Anxiety and anticipation of exercise are generally recognized as causes of inappropriate tachycardia with a corresponding diminution in stroke volume if cardiac output is not significantly increased. In spite of the increased variability which was found during standing, this is probably the only reasonable baseline condition for control determinations if the exercise is to be performed in the same position. The parallelism between dogs and human subjects is somewhat surprising since the trunk is horizontal in the standing dog.

SUMMARY

The ventricular chambers are frequently distended maximally while dogs and normal human subjects are relaxed and recumbent. Stroke volume also approaches maximal values under these conditions.
On sitting or standing, the heart diminishes and the ventricular stroke volume is reduced correspondingly in accordance with Starling's law of the heart.

The stroke volume increases but little during exertion and rarely exceeds recumbent control values. This increased stroke volume is generally achieved by increased systolic ejection, with little or no contribution by increased diastolic distention.

A distinction must be made between 3 baseline conditions during which control values are obtained in cardiovascular research: standing, recumbency, and after thoracotomy under anesthesia.

To evaluate cardiovascular effects of exertion while erect, it seems appropriate to obtain control values while standing quietly. Under these conditions, stroke volume is lower during the control than during exertion in most cases but not without exception. However, a progressive increase in stroke volume does not occur as the intensity of the exertion is increased.

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Summary in Interlingua

Le cameras ventricular es frequentemente distendite usque al maximo quando canes e normal subjectos human es relaxate e in postura recumbente. Etiam le volume per pulso approcha valores maximal sub iste conditiones.

In postura sedite o erecte, le corde se reduce e le volume per pulso ventricular ese reducete correspondentemente de acordo con le lege de Starling pro le corde.

Le volume per pulso se augmenta solmente un paucu sub conditiones de effortio e excede rarmente le valores de controlo pro le postura recumbente. Iste augmentate volume per pulso es generalmente effectuate per un augmento del ejection sistolica, con pauc o nulle contribution per un augmento del tension diastolic.

Un distinction debe esser facite inter 3 conditiones de base pro le quales valores de controlo es obtenite in recercas cardiovascular. Illos es le postura erecte, le postura recumbente, e le stato post-thoracotomy sub anesthesis.

Pro evalutar effectos cardiovascular de efforlio in postura erecte, il pare appropriate obtenere valores de controlo con le subjecto quiete in postura erecte. Sub iste conditiones le volume per pulso es plus basse in le condition de controlo que durant e le efforlio in le majoritate del casos sed non in omnes. Tamén, un augmento progressive del volume per pulso non occurre parallel al augmento del intensitate del effortio.

References

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The study of physiology and pathology within the past half-century has done more to emancipate medicine from routine and thraldom of authority than all the work of all the physicians from the days of Hippocrates to Jenner, and we are as yet upon the threshold.—William Osler, M.D. Medicine in the Nineteenth Century. New York Sun, 1901.
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