Orthostatic Factors in Pulsus Alternans

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Ventricular alternans was present in three cardiac patients only in the erect or semierect position and disappeared with recumbency. The phenomenon which was regularly reproduced in the standing posture was prevented or minimized by exercise, digitalis, infusions of blood and norepinephrine and by application of external vascular support. Ventricular alternation in the supine patient was noted only after phlebotomy, or venous pooling combined with Valsalva maneuvers. The observations suggest that in the mechanism of this type of pulsus alternans there is an important peripheral hemodynamic factor which exerts its effect by changes in diastolic length of an injured ventricular muscle.

In the past two years we have observed three patients with heart disease in whom pulsus alternans was strikingly related to posture. Ventricular alternans was present only in the erect or semierect position and disappeared with recumbency. The phenomenon could be reproduced at will, thus affording an opportunity to study some of the mechanisms concerned in its production.

The three subjects were considered to have varying degrees of organic heart disease. One person (J. W.) had hypertension with previous congestive heart failure, another (R. H.) had had congestive failure due probably to arteriosclerotic coronary artery disease, and the third individual had myocardial disease of uncertain etiology with an abnormal electrocardiogram and radiologic evidence of cardiac enlargement.

Case Reports

Case 1. J. W., a 55 year old Negro veteran had complained of various aches and pains in the back and extremities dating back to 1920. In 1935 he had an episode of pneumonia in the left lung; since then pains in the left anterior chest and subscapular region were added to his other skeletal pains. The pains were not specially related to effort, but he had not worked since 1935. He was then observed in numerous hospitals and treated for osteoarthritis of the spine. Dyspnea on exertion was present for 15 years. In February 1949, hypertension was noted for the first time at a level of 190/130. Persistent hypertension had been observed during the ensuing two years. In February 1950, he had increasing dyspnea and was given digitalis. In October 1950 he had stopped taking digitalis and developed paroxysmal dyspnea and mild pulmonary edema. In March 1951 he was admitted with principal complaints of head-aches, dyspnea, back ache and paresthesias. The significant findings at this time were blood pressure of 200/150, hypertensive retinopathy with marked narrowing of the retinal vessels, arteriovenous nicking and occasional exudate, and moderate cardiac enlargement. There was a grade II systolic blowing murmur at the base. The venous pressure was 13 cm. H₂O and the arm-to-tongue circulation time was 20 seconds. Pulsus alternans was present in the erect position, the difference in systolic pressure between the strong and weak beat amounting to as much as 30 mm. Hg. The electrocardiogram showed evidence of left ventricular hypertrophy. There was slight albuminuria and the maximum urine concentration was 1.020. The maximum urea clearance was 48 per cent and the phenolsulphonphthalein excretion was 5 per cent in 15 minutes. The excretory urogram showed an essentially normal configuration. The reaction to benzodiazepine injection was normal. The clinical impression was hypertensive cardiovascular disease and arteriolar nephrosclerosis.

Case 2. R. H., a 44 year old Negro man, was well until April 1949 when he first noted swelling of the ankles and dyspnea. The swelling progressed rapidly to the legs and abdomen. The dyspnea grew worse but was never prominent. During the ensuing two years he was observed in Veterans' Hospitals on three occasions. Each time he was found to have frank congestive heart failure with marked peripheral edema, pleural effusion, hepatic enlargement, elevated venous pressure and signs of pulmonary congestion, diffuse cardiac enlargement, gallop rhythm and markedly prolonged arm-to-tongue circulation time (35 to 55 seconds). On each occasion treatment with low salt diet, digitalis and diuretics effected prompt symptomatic improvement with loss of about 25 pounds of edema fluid and diminution in the size of the heart.

The etiology for the heart disease was not apparent. There had been no history of rheumatic fever or clinical signs of valvular disease. There were no evidences of syphilis by history, physical signs or laboratory tests. A blood pressure of 140/110 was...
present on one occasion during a period of severe failure. At other times the blood pressure readings were within the normal range (120/90, 120/92, 98/66). There was no history of previous hypertension or renal disease and no definite retinal vascular abnormalities. Although he was a moderate beer drinker, the dietary intake was considered to be adequate. There was no anemia. The determinations of the basal metabolic rates, when he was free of dyspnea, ranged between minus 9 and minus 19 percent, and the serum cholesterol values were slightly elevated (260 to 380 mg. per 100 cc.), but there were no other findings to suggest hypothyroidism.

Although he had not complained of chest pain, the electrocardiographic changes suggested the probability of coronary disease (absent R wave in V1 to V3, flat or inverted T waves in leads I, II, III, V4, and V5, and reversal of polarity of T wave in chest leads after exercise).

He was readmitted in February 1951. The break in compensation, like the one preceding, was precipitated by discontinuing digitalis and a low salt diet. In addition to the signs of congestive failure it was noted that he had definite pulsus alternans which was detected in the semierect or sitting posture, but was not apparent in the reclining position. A diffuse apical impulse was felt in the anterior axillary line in the left sixth intercostal space. The heart rate was 100, the rhythm normal and no murmurs were heard. Blood pressure was 120/90. Orthostatic pulsus alternans was still present in September 1953.

Case 3. E. B., a 48 year old Negro man, was admitted in July 1950, complaining of periubical and epigastric pain, nausea and vomiting of four days duration. A similar episode had occurred for the first time in April 1943, and required treatment in a military hospital. Since then he had had attacks of burning epigastric pain of a pattern resembling peptic ulcer. A transient syncopeal spell occurred in 1945 and was accompanied by dark stools. There was no additional history to suggest hemorrhage. In May 1948, he was treated at this hospital for uncomplicated duodenal ulcer. No clinical evidence of cardiac disease was noted at that time. Blood pressure was 128/90. An electrocardiogram was not taken. Fluoroscopic examination showed the heart and aorta to be normal. The blood serologic tests for syphilis were either negative or doubtful. There were no corroborative evidences of syphilis either by history or physical signs. There was no history of angina pectoris or of previous hypertension.

On admission in July 1950, he appeared well-nourished and developed and in no acute distress. There was slight, diffuse, midepiabdominal tenderness. There were varicose veins below the knee in both legs. The heart was not definitely enlarged. The heart rate was 110 per minute. In the sitting position every other pulse beat was weak and at times almost imperceptible. On assuming the reclining position the alternating pulse disappeared, and the rate declined slightly. The blood pressure reading, which initially was 150/106, declined shortly afterward to a level of 100/76. On subsequent examinations the blood pressure has usually been within normal range with occasional diastolic readings above 100 mm. Hg.

The pertinent laboratory data were as follows: Barium meal showed a clover-leaf deformity in the duodenum but no definite crater. The hemoglobin was 16.3 Gm., and repeated stool examinations were negative for occult blood. The serum chloride and bicarbonate were respectively 103 and 21 mEq. per liter. Fluoroscopic and x-ray examination of the chest revealed definite increase in size of heart over that noted two years previously. The electrocardiogram in the recumbent position was abnormal, showing diphasic T waves in leads I and II, deep Q3 and late deep inversion of T waves in V4, V5 and V6.

It was suspected at first that we were dealing with an acute myocardial infarction, and the patient was kept in bed for two weeks. The temperature, white blood count and sedimentation rate remained normal. The electrocardiogram did not show the progressive changes characteristic of an infarction. With management of the ulcer, the symptoms subsided. The phenomenon of pulsus alternans occurred only in the erect posture and was observed repeatedly at different intervals during the next 12 months.

The electrocardiographic changes and the increase in size of the heart persisted during the period of follow-up. The etiology of the heart disease was not clear. He had no symptoms of failing myocardial reserve and was not given digitalis.

Influence of Various Procedures upon Alternation

Effect of Body Position

The subjects reclined comfortably on a table which could be tilted to any desired angle. Records were made of the carotid or carotid-jugular pulse, electrocardiogram and heart sounds. Blood pressures in the arm were determined by means of a sphygmomanometer in the usual manner. When pulsus alternans was present, independent systolic readings could usually be registered for strong and weak beats. In some instances the electrokymogram and the femoral arterial pressure tracings were recorded.

In two subjects (R. H. and J. W., fig. 1) pulsus alternans was absent in the recumbent position and appeared first a few minutes after the body was tilted to an angle of 15 or 30 degrees from the horizontal. At a 60 degree angle the ventricular alternans was much more
Pulsus alternans was recorded in the erect posture at pulse rates varying from 70 to 140 per minute, but usually in the range of 90 to 110. The heart rates in all three subjects were faster in the vertical posture when pulsus alternans was present than in the horizontal position. The differences usually amounted to 10 to 30 beats per minute. Tachycardia of ectopic origin was never observed. The vertical posture was accompanied by minor alterations in blood pressure usually in the direction of a rise in the diastolic level and either no change or a slight rise in the systolic level. Postural hypotension was not present.

The electrocardiograph registered impulses of sinus origin and of identical configuration. Electrical alternans was not noted. Isolated beats of ectopic origin occurred sporadically and were then followed either by transient...

pronounced, the systolic level of the weak beat being about 20 mm. Hg below that of the strong beat of the couple. In the third individual (E. B., fig. 1) alternation of the beat could not be regularly induced except at an angle of at least 75 degrees, and then only after standing for five or six minutes. The longer he remained erect the more prominent it became. At times the weak beat produced an impulse too faint to be detected at the wrist. In all 3 persons tilting back into the horizontal position resulted in a prompt disappearance of the alternation.

These observations were repeated on numerous occasions during the ensuing year. Pulsus alternans was regularly present in the erect posture, varying only in degree and in the length of time required for the subject to remain standing before it became apparent.
accentuation of an existing ventricular alternation or by the induction of alternation for a period of six or eight beats.

Electrokymographic tracings obtained during periods of pulsus alternans in the erect posture showed concordant alternation in amplitude of ventricular contraction (fig. 5). This phenomenon was usually best recorded from aortic and left ventricular borders with the patient in the anteroposterior or left lateral roentgen position. An alternating pattern was not observed in tracings of the pulmonary arterial and auricular movements. As compared with the strong beat of the couple, the weak beat was characterized at its inception by a lesser level of diastolic filling and during contraction by an altered slope of systolic ejection and less complete emptying of the ventricle resulting in a fuller ventricular chamber (larger residual volume) at the end of systole.

**Effect of External Hydrostatic Pressure**

The effects of external hydrostatic pressure upon orthostatic alternation were observed while the patients were standing in a swimming pool filled with tepid water. Observations were begun as the subjects stood at the pool's edge, when alternation was marked. The subjects then descended by steps into the water, stopping for recordings on each step. In this manner the degree of external hydrostatic pressure was increased with the level of immersion from ankles to midchest.

It may be seen (fig. 2 A1–A5 B1–B5) that, as the water level rises above the hips, there is a gradual increase in the strength of the weak beat. Immersion to the midchest level abolished the alternation in one subject and nearly abolished it in the other for as long as the subjects stood in the water to this depth. Upon emergence from the water the tendency to alternation in strength of beat reappeared or became accentuated.

**Effect of Abdominal and Leg Binders**

To two patients in the supine position with the legs elevated above the head elastic bandages were applied firmly from the feet to the inguinal ligaments. Abdominal binders also were placed snugly in position. The subjects were then placed upright.

In each instance (fig. 2a) binding of the legs and abdomen prevented the development of pulsus alternans, which had unfailingly ap-
Effect of Exercise

Pulsus alternans was induced by placing the subject in the erect posture on the tilt table. He was then permitted to exercise in place for a period of 30 to 90 seconds by lifting the legs. Figure 3 represents the changes observed in typical experiments. In subject E. B. exercise resulted in a prompt disappearance of ventricular alternation despite an acceleration in heart rate of 20 beats per minute. In subject R. H. the strength of the weak beat was augmented after exercise but did not attain the full volume of the strong beat of the couple. Cessation of exercise was followed by rapid decline in amplitude of the weak beats prior to any appreciable change in heart rate.

Effect of Infusion of Blood and of Phlebotomy

In subject E. B. pulsus alternans was elicited with the patient on the tilt table at an angle of 78 degrees. The pulse rate fluctuated between 100 and 110 per minute, and the blood pressure was 110/75. Alternation was pronounced. The difference between strong and weak beat was more than 30 mm Hg, in fact, no systolic pressure was detected for the weak beat. Five hundred cc. of matched whole blood were infused over a period of 20 minutes. The weak beat became stronger after 300 cc. of blood had been infused, and at the end of the infusion period the pulse rate was 95 per minute and blood pressure 116/80. Pulsus alternans had entirely disappeared (fig. 4). During the ensuing 30 minutes, although the patient remained in the erect position, no alternation appeared except for four beats immediately following a premature ventricular systole.

On a different occasion, in the same individual an attempt was made to induce alternation of the pulse by means of phlebotomy while the patient was in the recumbent position. After 950 cc. of blood were withdrawn, the pulse rate increased from 87 to 107 per minute, but pulsus alternans was not present except for a transient period following immediately upon a premature beat. Prolonged alternation independent of premature beats appeared when the tilt table was elevated to an angle of 20 degrees from the horizontal, a position in which spontaneous ventricular alternation had never been present in this subject (fig. 1). While in this position alternation was stopped temporarily by light exercise, and completely when half of the previously removed blood volume had been reinfused.

At another time in this patient we did suc-
ceed in inducing temporary alternation in the completely recumbent posture. This was accomplished by means of venous occlusion tourniquets applied to all four extremities and the performance of a Valsalva maneuver at the same time (fig. 1).

Effect of Norepinephrine and Digitalis

Subject E. B. was resting on a tilt table in the erect posture at an angle of 80 degrees. The pulse rate was 108 and ventricular alternans was definitely present, the difference between strong and weak beats being about 12 mm. Hg \( \left( \text{blood pressure} \frac{130}{113} \right) \). Norepinephrine in saline, 4 micrograms per cubic centimeter, was infused intravenously for two minutes at a rate of 20 to 40 drops per minute. The pulse rate declined to 62 per minute simultaneously with a rise in blood pressure. When the blood pressure reached a level of 160/125 two minutes after beginning the infusion, pulsus alternans disappeared (fig. 4). Alternation reappeared within four minutes after stopping the infusion as the blood pressure returned to the preinfusion level. After a short rest period this experiment was repeated with identical results.

The effect of digitalization on orthostatic alternation was observed in subject E. B. who had never before been given digitalis in any
form. On this occasion, after having been standing for one hour, he had a pulse rate of 145 per minute, with marked pulsus alternans. In this position he was given lantoside C, 1.6 mg., intravenously. During the ensuing hour, although he remained standing, ventricular alternation tended to disappear for short intervals. One hour after the injection, by which time digitalis effects were present in the electrocardiogram, alternation disappeared for a 25-minute period. This reduction of orthostatic alternation was effected after digitalization, despite a concurrent increase in heart rate to 150 (fig. 4).

**DISCUSSION**

True ventricular alternation must be distinguished from pseudoalternans which may closely simulate it. The latter is observed often in bigeminy associated with periodically recurring premature ectopic beats or with sinoauricular or auriculoventricular conduction defects. It has been described in association with rapid breathing when the respiratory rate is half the pulse rate.1 Dyspnea was not present in the three patients here described at the time of these observations, and indeed most of the records were made with the patients holding the breath in the midrespiratory position. The electrocardiograms recorded during periods of pulsus alternans showed impulses of sinus origin and of similar cycle length and configuration for strong and weak beats with no variation in duration of P-R or Q-T intervals or QRS complex. In some records careful measurement disclosed a minute but periodic alternation in cycle length varying between 0.005 to 0.01 second but generally about 0.01 second in duration, the weak beat of the couple appearing closer in time to the antecedent than to the following strong beat. We have noted such slight periodic variations in cycle length in individuals with normal heart rates and true pulsus alternans unrelated to body position. A similar observation was made by Weunkebach.2

It is well known that a benign type of pulsus alternans may occur with disturbances of rhythm in an otherwise normal heart. Mechanical alternation of temporary duration has been observed following premature beats and for prolonged periods in association with marked tachycardia.3 4 Premature beats of ventricular origin were recorded in two of our three patients on several occasions. They were invariably followed in succession by a compensatory pause, a strong post-extrasystolic beat and either alternation of the pulse for six or eight beats or else an exaggeration of an already existing ventricular alternans for three or four couples. Ectopic beats never induced pulsus alternans of prolonged type. The latter was recorded in both subjects over periods of two or three hours without the appearance of a single ectopic beat.

The question arises whether the appearance of ventricular alternation in our cases is related solely to changes in heart rate. We do not believe this proposition to be valid for the following reasons: (1) The heart rates at which pulsus alternans appeared were frequently in the range of 80 to 90 per minute but varied widely in the same individual. (2) The differences in heart rate between the horizontal (nonalternating) posture and the vertical (alternating) position were small, being 10 to 30 beats per minute. (3) Following digitalis (fig. 4) and exercise (fig. 3) there was a lessening of the tendency to ventricular alternans in the face of no change or a slight increase in heart rate. While acceleration in heart rate undoubtedly exerts an important influence in the mechanism of pulsus alternans, we do not believe that the magnitude of the change here encountered constitutes the major factor in the induction of alternation in the erect position.

The patients described here have in common the existence of organic heart disease and pulsus alternans which is manifest in the erect or semirecumbent posture and absent in the recumbent position. The most obvious mechanism by which postural changes might influence the mechanical performance of the heart is by actions on the venous return, cardiac output and ventricular diastolic volume. The observations on these subjects and the postulated physiologic actions may be summarized as in table 1.

Those procedures which induced or amplified pulsus alternans curtail venous return and hence decrease ventricular diastolic volume.
Procedures which increase ventricular diastolic volume either by augmenting venous return or by increasing peripheral resistance tended to inhibit or abolish ventricular alternans. The favorable effect of digitalis in one case may be attributed to its action in improving efficiency of myocardial contraction so that increased work performance and presumably better emptying is accomplished with a shorter initial fiber length. A greater stretch, hence a more generous end-diastolic volume, is required to produce a strong beat. This end-diastolic volume is the sum of the residual volume just after systole plus the venous inflow during diastole. Under conditions of diminished diastolic filling, pulsus alternans comes about as follows: The weak beat fails to empty the ventricle, leaving a large residual volume; to this large residual volume is added the venous inflow, producing a large end-diastolic volume; because the end-diastolic volume is then adequate, a strong beat follows and empties the ventricle more completely; the remaining small residual volume plus the venous inflow leads to too small an end-diastolic volume for an optimum contraction, and a weak beat ensues.

The nearest approximation to the ventricular volume curve in our patients is the record of the left ventricular border movement as seen in the electrokymogram. The positional changes and artefactual movements that complicate the interpretations are admitted, but these objections are minimal in comparing consecutive beats in the same heart. In all of our kymographic tracings the strong beats begin at higher levels of diastolic volume than do the intervening weak beats and empty the heart more completely (fig. 5).

It is proposed that in these patients with diseased heart muscle the total contracting power is not sufficient to empty the ventricle normally under usual conditions of diastolic fiber length. A greater stretch, hence a more generous end-diastolic volume, is required to produce a strong beat. This end-diastolic volume is the sum of the residual volume just after systole plus the venous inflow during diastole. Under conditions of diminished diastolic filling, pulsus alternans comes about as follows: The weak beat fails to empty the ventricle, leaving a large residual volume; to this large residual volume is added the venous inflow, producing a large end-diastolic volume; because the end-diastolic volume is then adequate, a strong beat follows and empties the ventricle more completely; the remaining small residual volume plus the venous inflow leads to too small an end-diastolic volume for an optimum contraction, and a weak beat ensues.

The relative contributions of myocardial versus hemodynamic factors to the genesis of pulsus alternans has been a subject of discussion for more than 50 years. Wenekebach\(^2\) was the first to champion the concept that changes in ventricular filling and peripheral resistance may cause transient as well as sustained pulsus alternans quite apart from myogenic disturb-

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**Table 1.—Summary of Observations and Postulated Physiologic Actions**

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<thead>
<tr>
<th>Procedure</th>
<th>Physiologic Effect</th>
<th>Effect on Pulsus Alternans</th>
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</thead>
<tbody>
<tr>
<td>Erect posture</td>
<td>Diminish venous(^4,6) return and cardiac output</td>
<td>Induce or intensify</td>
</tr>
<tr>
<td>Recumbent posture</td>
<td>Increase venous return and cardiac output</td>
<td>Abolish or diminish</td>
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<tr>
<td>Exercise</td>
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<tr>
<td>Hydrostatic pressure</td>
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<tr>
<td>Elastic bandages</td>
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<tr>
<td>Increase blood volume</td>
<td></td>
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<tr>
<td>Nor-epinephrine</td>
<td>Increase peripheral resistance(^7) Improved myocardial efficiency(^8)</td>
<td>Abolish or diminish Abolish or diminish</td>
</tr>
<tr>
<td>Digitalis</td>
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</tbody>
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**Electrokymogram in Pulsus Alternans**

![Electrokymogram in Pulsus Alternans](image)

**Fig. 5.** Left ventricular electrokymogram in pulsus alternans. Time interval between solid vertical lines = 0.1 second.
ORTHOSTATIC FACTORS IN PULSUS ALTERNANS

ances. Wiggers\textsuperscript{3} supported this view with respect to the experimentally induced ventricular alternation which follows a prolonged diastolic pause. This type of alternation is temporary and occurs only in a rapidly beating heart. While all of the phenomena could be explained purely on the basis of hemodynamic alterations in his experimental preparation, Wiggers carefully emphasized the possibility of the importance of myocardial factors in other types of pulsus alternans.

Many investigators\textsuperscript{3, 10, 11} have emphasized the prime importance of variations in initial tension and volume in the mechanism of ventricular alternation. The point at issue has been whether the changes in volume and tension arise from primary alterations in vigor of muscle contraction or whether the latter are secondary to variations in inflow and resistance.

Greene\textsuperscript{12} demonstrated that pulsus alternans that follows experimentally induced coronary insufficiency is due to periodic deflections in contractile power of varying fractions of ischemic muscle in alternate beats. He believed that dynamic changes are not dominantly concerned in the mechanism of ventricular alternation since the degree of stretch in the weak beat was as great or greater than in the strong beat. His records show that the differences in degrees of stretch as registered by the myograph occur not at the end of diastolic filling but just prior to systolic ejection; during the period of isometric contraction. Lengthening of the muscle fiber of the localized area of injury at this point in the cycle reflects combined effects due to changes in diastolic volume plus changes in shape secondary to loss of contractile power. The greater fiber length in the weak beat may be interpreted as representing a greater degree of passive ballooning rather than increased stretch at the end of diastole. This evidence does not exclude the possibility of a significant hemodynamic factor.

Wiggers, a life-long investigator of this disturbance, recently summarized his views as follows: "It appears highly probable that ventricular alternation always involves the defection of some fractionate contractions during the small beat. However, changes in the intensity of alternation do not necessarily signify quantal variations in the defection of fractionate contractions. They can be induced by secondary dynamic factors which alter diastolic distention and initial tension."

The observations in patients with orthostatic alternation are in accord with this concept. Some procedures, such as exercise, augmented blood volume and acute hypertension, that abolished alternation in the standing subject can hardly have exerted their effect by improving myocardial contractability. Nor is it easy to visualize changes in intrinsic muscle contractile function arising as a result of immersing the body in water or of elevating the upper half 30 degrees.

We have not observed orthostatic alternation of the pulse in persons with normal hearts. Each of our three patients had evidence of diseased myocardium. In two it was severe enough to lead to congestive heart failure. Orthostatic hypotension was not present. One individual (E. B.) had prominent venous varicosities in the legs. He frequently displayed a rise in heart rate of 20 to 30 beats per minute on changing from the horizontal to the vertical position. External compression of the legs alone, whether applied by bandages or water, was not effective in entirely preventing the alternating phenomenon in this instance. The other two subjects had no detectable evidence of vasomotor instability or venous pooling.

We have observed three additional patients with heart disease and classic pulsus alternans which was present in the recumbent position but which was intensified on assuming the erect posture. It is, therefore, suggested that our three cases are not instances of an unusual vasomotor disturbance but rather accentuated forms of a phenomenon which may be common to all types of pulsus alternans.

We are not aware of any previous report in which the orthostatic posture is associated with pulsus alternans either to bring out a latent alternation or to exaggerate one which already exists.

It is evident that pulsus alternans may be completely overlooked in the recumbent subject. The patient who has been standing for
several minutes affords the optimum conditions for the detection of ventricular alternation.

**Summary**

1. Three patients with organic myocardial disease demonstrated the phenomenon of pul- sus alternans which was strikingly related to body position.

2. Measures which tended to induce or exaggerate alternation were erect posture, phlebotomy, venous pooling plus tourniquets and, for short periods, premature beats.

3. Procedures which tended to prevent or abolish ventricular alternations were recum- bency, exercise, digitalis, norepinephrine, trans- fusion of blood and application of external vascular support.

4. The observations support the view that there are two factors concerned in this type of alternation: (a) weakened or injured heart muscle which does not contribute sufficient contractile strength to empty the ventricle efficiently except under conditions of increased stretch; and (b) a precipitating extracardiac hemodynamic factor which exerts its effect by changes in ventricular inflow and peripheral resistance.

5. The detection of pulsus alternans may be facilitated if the patient is examined in the sitting or standing position.

**Sumario Español**

Alternans ventriculares se encontraron presentes en tres pacientes cardiacos en la posición vertical o semivertical pero desaparecieron con la reclinación. El fenómeno que fue reproducido con regularidad en la posición parada fué obviado o reducido al mínimo mediante el ejercicio, digitalis, infusiones de sangre y nor- epinefrina y mediante la aplicación de soportes vasculares externos. Alternación ventricular en el paciente supino se observó solamente luego de flebotomía o estancamiento venoso combinado con las maniobras de Valsalva. Las observaciones sugieren que en el mecanismo de este tipo de pulso alternans hay un factor hemodinámico periférico importante que ejerce su efecto por medio de cambios en el largo diástolico del músculo ventricular averiado.

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