Cardiocirculatory Studies in Pulsus Alternans of the Systemic and Pulmonary Circulations

By M. Irene Ferrer, M.D., Réjane M. Harvey, M.D., André Courmand, M.D., and Dickinson W. Richards, M.D.

Studies carried out in 21 patients have shown for the first time that alternation of the pulse pressure in man can occur independently in either the greater or the lesser circulation without appearing in the other. Even when bilateral alternation exists, this cyclic variation may disappear in one circulation while persisting in the other. Mechanisms responsible for pulsus alternans, in particular variations in stroke volume and vascular pressures, are considered, and no single explanation satisfies the facts revealed in this study.

Since Traube’s original description in 1872, the term pulsus alternans has been used to describe a special type of cyclic variation in systemic pulse pressure. Considerable clinical importance has since been attached to this circulatory sign, particularly since it is readily elicited with a simple sphygmomanometer. Although much laboratory investigation has been devoted to this subject, clinical exploration has been limited to studies of the systemic arteries in man until the advent of cardiac catheterization, when the exploration of both greater and lesser circulations could be made.

Prior to the present study, it had been more or less assumed that alternation in arterial pulse pressure was accompanied by alternation of both ventricular chambers simultaneously and synchronously. It is the purpose of this presentation to show, first, that alternation of the pulse pressure in man can occur independently in either the greater or the lesser circulation and, secondly, to demonstrate that the effects of digitalization, leg exercise, and variations in vascular pressures upon pulsus alternans have once more raised questions regarding the mechanisms responsible for this dynamic cyclic event.

Over the past few years, during an investigation of a variety of circulatory abnormalities, pulsus alternans was encountered in 21 patients with cardiovascular disease, although a specific search for this abnormality was not being made. The observations secured in this group form the basis of this paper. The cardiac catheterization technic, utilizing the Fick principle for determination of the cardiac output was employed, and Hamilton manometers or strain-gage pressure transducers with a photo-oscillographic technic* supplied the pressure curves.

Definition of Pulsus Alternans

The cyclic event termed pulsus alternans in this presentation refers to a circulatory state in which there was a regular alternation in pulse pressure, accompanied by a regular rhythm without appreciable variation in cardiac cycle length. The rhythm was of sinus origin in 19 of the 21 cases while atrial flutter and supraventricular tachycardia, probably of A-V nodal origin, were present respectively in each of the remaining 2 cases. It should be emphasized that electric alternans, i.e., change in the size of the electrocardiographic QRS complex, was not present in any of the cases under discussion.

* The pressure-recording machine was manufactured by Electronics for Medicine, Inc., White Plains, N. Y.
Fig. 1. Pulsus alternans in brachial artery. Pressure curves depicting pulsus alternans in the brachial artery (B.A., upper curve) in patient H.I. no. 345. The right atrial (R.A.) curve below the latter shows no alternation. The scale in mm. Hg for each curve appears to the left and standard lead II appears at the bottom of the frame. The maximum systolic difference between the large and small beats in the brachial artery is 5 mm. Hg. These curves were recorded at rest using Hamilton manometers. In this and subsequent figures, unless otherwise noted, the time lines are 0.04 sec. apart and a bar encloses an interval of 0.20 sec.

An example of the classical features of pulsus alternans appears in figure 1, where the brachial artery pressure curve is characterized by an alternately large and small beat.

*False Alternans.* Instances classed as false alternans were carefully excluded from this study. These are produced either by variation in cardiac cycle length or the effects of breathing, which, respectively, alter diastolic filling time and venous inflow. Some examples of false alternans are found in figures 2 and 3. False alternans can also be produced in the presence of regular rhythm if respiratory and ventricular rates bear a 1:2 or 1:3 relationship to each other.

*True Alternans.* To return to true pulsus alternans, perhaps the most interesting fact uncovered in this study is the demonstration of the independent alternation of systemic artery or pulmonary artery, a fact previously unknown in clinical medicine. This may be seen in figure 4, where alternation occurs in the brachial artery only, with none in the pulmonary artery or right ventricle. In addition, it is obvious that in this instance alternation occurs in the systolic phase alone, while the diastolic brachial pressure varies only with the respiratory cycle as occurs normally. Figure 5 presents an alternating pulmonary artery; again the variation is apparent only in systole, without evidence of alternans on the systemic side, where there is only a gradual respiratory waxing andwaning of the pulse pressure in the femoral artery. It could be argued that diastolic alternation in the pulmonary artery was obscured by the marked effects of respiration upon this pulse contour and, indeed, as can be seen on the left of figure 6, these respiratory effects can almost obliterate even the systolic alternation at times. In the right side of the same figure, recorded when the breath was held, systolic alternans comes out clearly and no diastolic alternans is discernible.

Alternation of pulse pressure in both great vessels is illustrated in figure 7. It is quite obvious that the alternation occurs synchronously in both sides of the circulation, as was true in the other 8 cases with bilateral alternans in this series. These pressures were always recorded simultaneously in contradistinction to the case of DeRabago, where they were not simultaneous, and in whom a nonsynchronous alternation was postulated. In contrast to the previous figures, alternation in figure 7, which was recorded with the breath held, involves both the systolic and diastolic levels in these arteries.

The coexistence of alternation in both circulations is not necessarily fixed, since, as will be seen in the next 3 illustrations, disappearance of pulsus alternans is not necessarily simultaneous in both circuits. In the first illustration (fig. 8), one notes the initiation of bilateral alternans after only 1 premature atrial contraction. Although alternation persists for the 10 beats in the pulmonary artery between the arrows in figure 8, alternans in the brachial artery ceases after the fourth postextrasystolic beat. It is well known that the appearance of variations in size of pulse beats after a premature contraction can persist for 2 or 3 beats after the premature systole on the basis of readjustments in stroke volume. It is difficult to believe that such adjustments would go on for as long as 10 beats. At all events, it would not explain the persistence of alternans in the pulmonary artery when it had ceased in the brachial artery. Figure 9 demonstrates alternans bilaterally in the brachial artery and right
Fig. 2. False alternans during sinus arrhythmia. Pressure curves depicting false alternans in both lesser and greater circulations during sinus arrhythmia (N.G. no. 770). The upper curve is from the pulmonary artery (P.A.) and the lower from the brachial artery (B.A.), both with scales to the left. Lead II appears at the bottom. These curves, and those in figures 4-7, 11, and 13 were recorded by the use of strain-gage pressure transducers and a photo-oscillographic technic. Note that the pulmonary artery shows the alternating size of the pulse waves more clearly than the brachial artery below it. The diastolic pressure level varies directly with the respiratory cycle, as is the case normally, reaching its peak at full inspiration. The pulse pressure of these beats, however, is related to long and short cycle times as well as to respiratory variations. The second and third pulmonary artery beats have the same cycle lengths but occur during the inspiratory phase, hence the second exceeds the first in height, while the third is waning. The fourth beat, however, occurs after a longer diastole and although from the respiratory pattern it should be declining, it is larger, due to a longer filling time, a greater diastolic volume, and presumably a larger stroke volume. The fifth beat should be the smallest owing to the respiratory influence, but having the longest diastole of all, hence the largest stroke output, it achieves the same size as the fourth beat. The sixth beat with a short diastole occurs at the end of expiration and is again a small one, while with inspiration the seventh beat grows larger despite a short filling period. Here then is a nice balance shown between the influence of respiration on the one hand and cycle length or filling time on the other.

Fig. 3. False alternans in brachial artery due to atrial premature contractions. Pressure curves depicting false alternans in the brachial artery (B.A.) due to the regular recurrence of atrial premature contractions (I.J. no. 430). Note the usual small beat in the brachial artery following the premature excitation, the larger postextrasystolic beat and the smaller beat that follows the latter. The timing of the premature atrial contractions is such that, because of variations in diastolic filling and ventricular emptying, a continuous alternation in the brachial artery occurs until the last 5 beats in the figure, where normal sinus rhythm appears uninterrupted by atrial premature systoles. The atrial curve (R.A.) shows a mechanical atrial event for each premature beat (APC) but no alternation. Lead II is recorded at the bottom of the frame. The series of arrows indicate the registration of the atrial systoles, including 1 premature atrial beat, on the brachial artery curve. Hamilton manometers were used.
ventricle and indicates that after quinidine this latter chamber no longer alternates. The drug produced a fall in right ventricular pressure but effected no change in cardiac output. The cyclic variation in pulse pressure however persists in the brachial artery. Finally, as shown in figure 10, during acute digitalization of a patient in congestive failure with bilateral alternans, the alternate fluctuation in the right ventricle disappears 10 minutes after digoxin was given intravenously, at a time when the cardiac output had risen and the pulmonary artery and right ventricular systolic and diastolic pressures had declined, i.e., when presumably both left and right myocardial performance had been improved. The brachial artery alternans, on the other hand, did not vanish for another 30 minutes. These 3 figures therefore demonstrate the conversion of bilateral into unilateral pulsus alternans. This fact stresses the relatively independent behavior of each of the 2 ventricles and their respective circulations.

The data so far presented have emphasized the existence of unilateral and bilateral pulsus alternans. It has been shown that certain cardiac drugs can obliterate this cyclic dynamic feature in one or the other circulation. Further-

Fig. 4. Pulsus alternans in brachial artery alone. Pressure curves depicting pulsus alternans in the brachial artery alone. This was recorded in the recovery period, 15 min. after exercise was stopped, in patient D.C. no. 778. The upper curves are from the pulmonary artery (P.A.) and right ventricle (R.V.) and the lower from the brachial artery (B.A.) with lead II at the bottom. The maximum systolic difference between large and small beats is 20 mm. Hg. See text for discussion.

Fig. 5. Pulsus alternans in pulmonary artery alone. Pressure curves depicting pulsus alternans in the pulmonary artery alone (A.H. no. 833). The upper curve is from the pulmonary artery (P.A.) and the lower from the femoral artery (F.A.) with lead II recorded between them. The maximum systolic difference between large and small beats is 9 mm. Hg. See text for discussion.
more, a temporary imbalance in the regularity of stroke output as induced by 1 premature systole, was shown to be capable of setting off alternation, a phenomenon that has been described by other investigators.\textsuperscript{1} \textsuperscript{2} These latter considerations immediately raise the question of the mechanism that may be involved in the production of pulsus alternans. Before considering such mechanisms, certain clinical features associated with pulsus alternans deserve mention.

**CLINICAL FEATURES**

The relationship between the etiology of the heart disease in these subjects and the presence of unilateral and bilateral pulsus alternans, as well as other related clinical and hemodynamic findings, are of interest and can be seen in table 1. In this series of 21 cardiac patients whose age ranged from 32 to 69 years, 18 must be considered to have advanced heart disease. Three patients (no. 447, 778, 782) however, were minimally, if at all, limited by their disease. Six patients showed alternans on the pulmonary side alone; they suffered from hypertensive or rheumatic heart disease, and 1 had chronic cor pulmonale. Five of these 6 had moderate to severe pulmonary hypertension, while in 1, pulmonary artery pressures were normal during alternation. The latter fact suggests that the congested state is not always a necessary accompaniment of pulsus alternans. Six other patients alternated only on the systemic side of the circulation and, of these, 3 were hypertensive while arteriosclerotic or valvular heart disease made up the remainder. Seven of the 9 patients with bilateral alternans had hypertensive or arteriosclerotic disease, or both, and 1 was considered to have idiopathic ventricular hypertrophy, while in the ninth the etiology was unknown. In this last group with bilateral alternans, 8 of the 9 patients were in

---

**Fig. 6.** Pulsus alternans in pulmonary artery. Pressure curves depicting pulsus alternans in the pulmonary artery alone during breathing and with breath held (A.H. no. 833). Curves are from the same patient and are arranged as in figure 5. See text for discussion.

**Fig. 7.** Pulsus alternans in brachial and pulmonary artery. Pressure curves depicting pulsus alternans in brachial (B.A.) and pulmonary arteries (P.A.) (B.Y. no. 949). The maximum systolic difference between large and small beats is the same in both arteries, 13 mm. Hg. Lead II appears at the top. See text for discussion.
congestive right and left heart failure with pulmonary and right ventricular hypertension, whereas only one half of each of the groups with unilateral alternans had congestive failure. The degree of alternation on the systemic side varied by as little as 5 and by as much as 30 mm. Hg, while alternating systolic peaks in the lesser circulation differed by 5 to 13 mm. Hg. The cardiac output during alternation in these subjects was either normal or reduced.

A clinical point that has impressed the authors is the almost constant occurrence of an apical diastolic gallop whenever systemic pulsus alternans was found. This relationship was followed by means of a stethogram during the acute digitalization of 1 patient and when the bilateral alternans had disappeared the gallop too had been lost.

The association of intraventricular conduction defects with pulsus alternans is not a close one, since, as can be noted in table 1, bundle-branch block existed in only 3 patients (no. 734, 477, 306). Two (no. 734, 477) had right bundle-branch block and alternated in only the greater or in both circulations. The conduction defect was on the left in the third patient (no. 306) as was the alternans. It was in only this 1 individual (no. 306), who has been described in a previous paper, that the time relationship between the onset of the QRS complex and the onset of the systolic pressure rise in the femoral artery varied, the Q-FAs time being longer for
the smaller beat. Whether this alternate lengthening of the Q-FA interval stems from a prolongation of isometric contraction or a decrease in pulse-wave velocity is not known.

**Discussion**

In considering mechanisms of pulsus alternans, one of the classic concepts that grew out of the animal work on this subject stresses the question of variations in stroke volume. There is no way in which stroke output of each ventricle can be assessed directly in man, but an indication of possible variations in stroke output, if one accepts and applies the Frank-Starling Law, can be had by examination of the diastolic ventricular pressure levels of the individual beats. The pulsus alternans induced in animals has been found to include variations in this diastolic pressure between the small and large beats as well as variations in stroke volume, and it was therefore assumed that this diastolic fluctuation was an integral part of the alternating ventricular systoles.

Examination of the pressure curves obtained in a patient with unilateral right sided pulsus alternans provides information relative to this question of alternation in diastolic pressure levels. Figure 11 shows a right ventricular curve showing ventricular alternans recorded while the patient was holding his breath, a maneuver that minimizes changes in venous inflow as well as variations in pleural pressure, both of which can influence diastolic pressure. It is clear in this figure that only the systolic level alternates, the diastolic pressure remaining at the same level for the large as well as the small beat. Since there is no fluctuation in diastolic filling time as cycle lengths are equal, and no variation in diastolic ventricular pressure, diastolic volume changes must be minimal if present at all. It is also interesting that in none of the patients in this series was alternation found in the right atrial curve.

It is appropriate to note in connection with diastolic filling time, that heart rate was variable in these subjects, and right or left alternans
### Table 1.—Clinical and Physiologic Findings in Twenty-one Patients with Pulsus Alternans

The values given in this table were obtained under resting basal conditions. Unless coupled with a second figure in parentheses, they represent the values obtaining while alternation was present. In four subjects where resting alternans was not present but where alternans appeared in relation to exercise, and in one subject where sudden change in heart rate was associated with alternans, the values during alternation appear in parentheses.

<table>
<thead>
<tr>
<th>Case, age (yr.), sex</th>
<th>Diagnosis</th>
<th>ECG findings</th>
<th>Cardiac index (L/min./M² RHS)</th>
<th>Heart rate</th>
<th>Pressures in mm. Hg</th>
<th>TBV (ml./M² RSA)</th>
<th>PV (ml./M² RSA)</th>
<th>Hematocrit (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Systemic artery s/d, m</td>
<td>Pulmonary artery s/d, m</td>
<td>Right ventricle s/d</td>
<td></td>
</tr>
<tr>
<td>*334, T.C. 49♂</td>
<td>CPE, CCP, EH, (G-S), RVF</td>
<td>NST, READ</td>
<td>3.18</td>
<td>110</td>
<td>115/74, 89</td>
<td>—</td>
<td>110/7</td>
<td>4100 1810 57</td>
</tr>
<tr>
<td>*609, J.B. 34♂</td>
<td>*RHD, EH, MS</td>
<td>NSR, READ, RVH</td>
<td>1.83 (E 1.73)</td>
<td>98  (E 142)</td>
<td>98/68, 85</td>
<td>117/61, 87</td>
<td>—</td>
<td>2880 1545 46</td>
</tr>
<tr>
<td>*723, F.H. 37♂</td>
<td>*RHD, EH, MS, MI, (G-S), CHF</td>
<td>NST, READ, RVH</td>
<td>1.56</td>
<td>107</td>
<td>130/83, 99</td>
<td>86/55, 67</td>
<td>88/15</td>
<td>2171 1583 59</td>
</tr>
<tr>
<td>*833, A.H. 39♀</td>
<td>RHD, EH, MS</td>
<td>NST, READ, RVH</td>
<td>2.87</td>
<td>115</td>
<td>114/69, 98</td>
<td>63/48, 68</td>
<td>93/3</td>
<td>2094 1149 47</td>
</tr>
<tr>
<td>*856, C.G. 57♂</td>
<td>HCVD, EH, CHF</td>
<td>NSR, NEAD</td>
<td>2.03</td>
<td>62</td>
<td>208/125, 157</td>
<td>76/44, 60</td>
<td>76/14</td>
<td>4150 1816 55</td>
</tr>
<tr>
<td>*739, M.C. 66♀</td>
<td>HCVD, ASHD, EH, CS, MF, OMI, CHF</td>
<td>NSR, LEAD, Low voltage, QrT pattern</td>
<td>1.02</td>
<td>69</td>
<td>148/71, 102</td>
<td>25/9, 15</td>
<td>25/1</td>
<td>2572 1543 40</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Case, age (yr.), sex</th>
<th>Diagnosis</th>
<th>ECG findings</th>
<th>Cardiac index (L/min./M² RHS)</th>
<th>Heart rate</th>
<th>Pressures in mm. Hg</th>
<th>TBV (ml./M² RSA)</th>
<th>PV (ml./M² RSA)</th>
<th>Hematocrit (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Systemic artery s/d, m</td>
<td>Pulmonary artery s/d, m</td>
<td>Right ventricle s/d</td>
<td></td>
</tr>
<tr>
<td>*306, S.H. 57♂</td>
<td>HCVD, ASHD, EH, CS, MF, CHF</td>
<td>NSR, LEAD, LBBB with Q.</td>
<td>2.03</td>
<td>74</td>
<td>196/108, 135</td>
<td>—</td>
<td>33/7</td>
<td>2910 1760 40</td>
</tr>
<tr>
<td>*447, E.B. 69♀</td>
<td>ASHD, CS, MF</td>
<td>NSR, NEAD</td>
<td>3.85</td>
<td>85</td>
<td>184/82, 118</td>
<td>24/8, 12</td>
<td>24/0</td>
<td>2880 1750 49</td>
</tr>
<tr>
<td>*778, D.C. 42♀</td>
<td>HCVD, EH</td>
<td>NSR, LEAD (lat. study)</td>
<td>3.62 (E 4.27)</td>
<td>88  (E 120)</td>
<td>283/118, 181</td>
<td>37/18, 28</td>
<td>37/4</td>
<td>2635 1342 49</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(2nd. study)</td>
<td>3.24</td>
<td>E 4.38</td>
<td>209/133, 171</td>
<td>E 61/31, 46</td>
<td>—</td>
<td>6073 1498 50</td>
</tr>
<tr>
<td>*477, W.W. 56♂</td>
<td>ASHD, EH, CS, MF, CHF</td>
<td>Atrial flutter, RBBB</td>
<td>1.85</td>
<td>200</td>
<td>106/81, 87</td>
<td>22/15, 17</td>
<td>22/9</td>
<td>3900 2110 46</td>
</tr>
<tr>
<td>*782, L.L. 32♀</td>
<td>RHD, EH, MS, MI</td>
<td>NST, NEAD</td>
<td>3.23</td>
<td>E 5.66</td>
<td>126/70, 91</td>
<td>18/11, 14</td>
<td>18/2</td>
<td>2307 1375 42</td>
</tr>
<tr>
<td>*788, J.D. 55♂</td>
<td>*CAD, EH, AS, AI</td>
<td>NSR, NEAD</td>
<td>3.25 (E 5.36)</td>
<td>88  (E 132)</td>
<td>135/54, 86</td>
<td>20/7, 14</td>
<td>20/0</td>
<td>3131 2002 36</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Case, age (yr.), sex</th>
<th>Diagnosis</th>
<th>ECG findings</th>
<th>Cardiac index (L/min./M² RHS)</th>
<th>Heart rate</th>
<th>Pressures in mm. Hg</th>
<th>TBV (ml./M² RSA)</th>
<th>PV (ml./M² RSA)</th>
<th>Hematocrit (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Systemic artery s/d, m</td>
<td>Pulmonary artery s/d, m</td>
<td>Right ventricle s/d</td>
<td></td>
</tr>
<tr>
<td>*466, G.R. 54♂</td>
<td>HCVD, EH, LVF</td>
<td>NST, LEAD</td>
<td>2.20</td>
<td>120</td>
<td>215/144, 166</td>
<td>57/43, 48</td>
<td>57/3</td>
<td>3140 1740 45</td>
</tr>
<tr>
<td>*345, H.I. 81♂</td>
<td>HCVD, EH, CHF</td>
<td>NST, LEAD</td>
<td>2.77</td>
<td>110</td>
<td>222/130, 162</td>
<td>—</td>
<td>60/9</td>
<td>3300 1775 44</td>
</tr>
<tr>
<td>*519, J.M. 38♂</td>
<td>HCVD, EH, CHF</td>
<td>NST, LEAD</td>
<td>2.15</td>
<td>111</td>
<td>210/131, 155</td>
<td>57/30, 40</td>
<td>57/11</td>
<td>3340 1940 42</td>
</tr>
</tbody>
</table>
Table 1.—Continued

| Case, age (yr.), sex | Diagnosis | ECG findings | Cardiac index (L/min./M² BSA) | Heart rate | Pressures in mm. Hg | Hematocrit (%) |
|---------------------|----------|--------------|-------------------------------|------------|---------------------|----------------|----------------|
|                     |          |              |                               |            | Systemic artery s/d, m | Pulmonary artery s/d, m | Right ventricle s/d | TBV (ml./M² BSA) | PV (ml./M² BSA) |
| #615, W.M. 35 ♂     | HCVD, EH, CHF | NSR, NEAD | 2.56                          | 98         | 173/123, 140         | 56/25, 39         | 56/10          | 3237           | 2145           | 33             |
| #602, M.R. 63 ♂     | ASHD, EH, CS, MF | NST, NEAD (Supravent. tachycardia) | —           | 103          | 125/99, 99 (135/76, 99) | 43/13, 28 (25/10, 18) | 43/4           | 3250           | 1925           | 41             |
| #670, A.S. 67 ♂     | ASHD, EH, CS, MF, CHF, CPE | NST, LEAD | —                            | 111         | 145/92, 102         | 57/23, 35         | 57/11          | 3240           | 1867           | 47             |
| #685, A.R. 46 ♂     | IVH, EH | NSR, NEAD, LVH | 1.94                         | 100         | 128/85, 102         | 21/11, 14         | —              | 3340           | 1770           | 50             |
| #734, L.K. 53 ♂     | UHD, EH, CHF | NSR, READ, RBBB | —                            | 100         | 110/74, 92          | 66/34, 47         | 66/15          | —              | —              | —              |
| #949, B.Y. 60 ♂     | HCVD, ASHD, EH, CS, MF, CHF | NST, LEAD | 1.79                         | 117         | 152/05, 116         | 75/40, 53         | 75/15          | 2975           | 1607           | 43             |

* Confirmed by necropsy; AI = aortic insufficiency; A2 = aortic stenosis; ASHD = arteriosclerotic heart disease; CAD = calcific aortic disease; CCP = chronic cor pulmonale; CHF = congestive heart failure; CPE = chronic pulmonary emphysema; CS = coronary sclerosis; E = exercise; EH = enlarged heart; (G-S) = Graham-Steell murmur; HCVD = hypertensive cardiovascular disease; IVH = idiopathic ventricular hypertrophy; LBBB = left bundle-branch block; LEAD = left electric axis deviation; LVF = left ventricular failure; LVH = left ventricular hypertrophy; MF = myocardial fibrosis; MI = mitral insufficiency; MS = mitral stenosis; NEAD = no electric axis deviation; NSR = normal sinus rhythm; NST = normal sinus tachycardia; OMI = old myocardial infarct; PV = plasma volume; R = recovery; RBBB = right bundle-branch block; READ = right electric axis deviation; RHD = rheumatic heart disease; RVF = right ventricular failure; RVH = right ventricular hypertrophy; TBV = total blood volume; UHD = unknown heart disease.

Fig. 11. Right ventricular alternation with breath held. Pressure curves depicting right ventricular (R.V.) alternation only and recorded with the breath held (A.H. no. 833). The maximum systolic difference between large and small beats is 6 mm Hg. The femoral artery (F.A.) curve is at the bottom and lead II runs through the right ventricular curve. See text for discussion.
Alternans was found during exercise (no. 788) at a time when systemic pressures had risen. No alternation appeared, however, in the lesser circuit although pulmonary pressures increased sharply. In another subject (no. 778, first study), systemic alternans, although it began during exercise, became more marked in the recovery period when pressures had fallen (fig. 13). In contrast, systemic alternans has also been noted only in the recovery period in
2 individuals (no. 778, second study, and no. 782) when pressures that had previously risen during exercise were no higher than at rest. Finally, figure 14 presents a femoral artery alternation that disappeared when this hypertensive patient’s blood pressure fell during a vasovagal episode. It is obvious from the widely divergent fluctuations in vascular pressures here described that there is no invariable relationship between changes in pressure and the appearance or disappearance of pulsus alternans. Whether such pressure variations indicate variations in resistance, cannot be stated.

CONCLUSION

The rhythmicity of the phenomenon of pulsus alternans has fascinated physiologists and clinicians for many years and there is still doubt as to its genesis. Wiggers\(^5\) summarizes his views by saying that probably ventricular alternation always involves periodic deflections in contractile power of the myocardial fibers, although he admits the possibility of secondary dynamic factors. It is possible that cyclic variations in myocardial contraction can be limited only to those fibers over 1 or the other ventricular chamber in unilateral pulsus alternans. However, if the small beat is really the weaker beat, and this is taken to mean that the small beat delivers the smaller stroke volume, then one would expect, if the Frank-Starling Law applies in these conditions, at least some evidence of alternating end-diastolic ventricular pressure. This has not been found in our patients. Possibly, of course, such pressure changes were too small to be detected in the lesser circulation by present methods in man.

If variable fractionate contractions of the myocardium do not constitute the sole regulating mechanism of this remarkably regular waxing and waning in pulse pressure, one is forced to look for additional and perhaps as yet undiscovered factors. Obviously, variations in pressure and possibly resistance may affect pulsus alternans, but the mechanisms involved are not simple and are probably allied in some fashion with myocardial performance. It may be that there is a resonance phenomenon in the entire ventricular-vascular system by which these alternate systolic amplitudes are initiated and sustained. Be that as it may, unilateral alternans may offer the opportunity in the future of examining in greater detail in man the regulatory factors concerned in producing this interesting phenomenon.

SUMMARY

Studies carried out in 21 patients have indicated that alternation of the pulse pressure in man can occur independently in either the greater or the lesser circulation without appearing in the other. Even when bilateral alternation exists, this cyclic variation may disappear in one circulation while persisting in the other.

Although pulmonary or systemic hypertension was frequently associated with pulmonary and systemic alternans respectively, hypertension was not invariably present. Furthermore, changes in lesser or greater circulation pressures bore no consistent relationship to the appearance or disappearance of pulsus alternans.

Mechanisms responsible for pulsus alternans, in particular variations in stroke volume and vascular pressures, were considered, but no single explanation satisfied the facts revealed in this study.

SUMMARIO IN INTERLINGUA

Studios in 21 patientes ha indicate que alternation del pression pulsati pote occurrer in homines de manera independente in o le
circulation major o le circulation minor. Mesmo in casos de alternation bilaterale, iste variation cyclic pote disperar in un del circulationes e persistir in le altere.

Ben que hypertension pulmonar o systemic esseva frequentemente associate con pulmonar e systemic pulso alternante, respectivamente, le presentia de hypertension non esseva sin exception. In plus, alterationes del pression in le circulationes major e minor non esseva regularmente relationate al apparition o disparition de pulso alternante.

Nulle specific explication del factos revelate in le presente studio se provava satisfactori ben que varie mechanismos responsabile pro pulso alternante esseva prendite in consideration—specialmente variationes del volumine per pulso e del pressiones vascular.

REFERENCES


Medical Eponyms

By Robert W. Buck, M.D.


In his conclusion he says (p. 419):

“...The blue sickness, especially when diagnosed in the adult, is the result of a small number of perfectly definite cardiac malformations.

“Of these cardiac malformations, one exceeds all the others in frequency, since we have found it in nearly 74 per cent of our cases. . . .

“This malformation is a true anatomical-pathological entity, represented by the following tetralogy: (1) stenosis of the pulmonary artery, (2) communication between the ventricles, (3) displacement to the right of the origin of the aorta, (4) hypertrophy of the right ventricle, almost always concentric in type. To these may occasionally be added, with only accessory significance, a persistent ductus Botalli.”
Cardiocirculatory Studies in Pulsus Alternans of the Systemic and Pulmonary
Circulations
M. IRENÉ FERRER, RÉJANE M. HARVEY, ANDRÉ COURNAND and DICKINSON W. RICHARDS

Circulation. 1956;14:163-174
doi: 10.1161/01.CIR.14.2.163
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1956 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/14/2/163

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/