The Influence of Advanced Congestive Heart Failure on Pulsus Alternans

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Evidence is presented which indicates that in certain instances pulsus alternans may be abolished or diminished in the presence of advanced congestive heart failure. The observations suggest that in severe congestive failure the ventricular filling pressure may increase to the point where it causes the diastolic stretch of the left ventricle to be more equal from one beat to the next. This in turn would bring about more uniform systolic ejections and hence ventricular alternation would disappear or be lessened in degree.

Pulsus Alternans is considered a grave sign of heart disease, indicating myocardial insufficiency. Recent observations indicate that this phenomenon can be profoundly influenced by changes in the venous return to the heart. During the past two years we have encountered 12 patients with pulsus alternans and have noted that in certain instances advanced congestive failure may also influence pulsus alternans. We wish to report detailed studies of three of these patients.

Material and Methods

Three patients, all of whom showed pulsus alternans initially and who were on accepted treatment for cardiac failure were observed. One, who had rheumatic aortic insufficiency, was in heart failure following treated subacute bacterial endocarditis. Two observations were made on this man; the first, when he showed early signs and symptoms of left heart failure, and the second, when he was in intractable congestive failure seven months later. The remaining two patients had hypertensive heart disease with severe congestive failure as evidenced by dyspnea at rest, orthopnea, paroxysmal nocturnal dyspnea, venous distention, liver engorgement and peripheral edema. After the pulsus alternans was demonstrated, both were taken off all treatment for heart failure and were put on an unrestricted sodium diet for a period of 7 to 10 days. They were followed both symptomatically and by weight. Repeated observations were made of the arterial pulse. Treatment was reinstituted at the end of this period and again the pulse was recorded. All pulse curves shown were taken after the patient had been standing upright for a period of 20 to 30 minutes.

The direct arterial pulse was recorded simultaneously with the electrocardiogram and pneumogram, using string galvanometers. A Statham pressure transducer with a calibrated control box was used for the pulse curves.

Results

Representative pulse curves of these three patients are seen in figures 1, 2 and 3.

Figure 1 shows C. B., a 29 year old white male, with rheumatic aortic insufficiency. When early heart failure was manifest, an alternans of 12 mm. was present. Seven months later when intractable congestive failure was present, the alternans had disappeared.

Figure 2 shows W. J., a 42 year old Negro man, with hypertensive heart disease. At the time of admission an alternans of 6 to 8 mm. was present. At that time he weighed 235 pounds. Off treatment for one week with a progression of symptoms and a weight gain of 5 pounds, the alternans had virtually disappeared. One week after treatment was restarted, the patient had improved symptomatically and had a weight loss of 28 pounds. Weighing 212 pounds, an alternans of 8 to 10 mm. was present. Two and one half months after treatment was restarted further improvement was noted. His weight was 185 pounds, and he was then edema free. An alternans of 16 to 18 mm. was seen.

Figure 3 shows A. R., a Negro woman, aged

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FIG. 1. C. B., a 29 year old white man with Rheumatic Heart Disease with Aortic Insufficiency. (a) At the time of early failure while on treatment; (b) At the time of severe intractable failure while on treatment. This and the following curves are reproductions of the actual pulse tracings.

FIG. 2. W. J., a 42 year old Negro man with Hypertensive Heart Disease. (a) At the time of admission while on treatment; (b) One week after treatment discontinued; (c) Five days after treatment restarted; (d) Seven weeks after treatment restarted.

FIG. 3. A. R., 45 yr. old Negro woman with Hypertensive Heart Disease. (a) On admission while on treatment; (b) Nine days after treatment discontinued; (c) Two months after treatment restarted. Note the reduced sensitivity of curves (b) and (c).

45, with hypertensive heart disease. Two days after admission, the weight was 148 pounds, and an alternans of 10 mm. was noted. Ten days after treatment was discontinued, there was a weight gain of 4 pounds with progression of symptoms. Less than 5 mm. of alternation was demonstrable. Eight weeks after treatment was reinstituted the patient was improved. She then weighed 140 pounds although some edema was still present. An alternans of 14 mm. Hg was noted.

The results are summarized in table 1.

It might be pointed out that it has not been our experience thus far, for pulsus alternans to be exaggerated as congestive failure becomes more severe.

DISCUSSION

Our observations do not explain the actual cause of pulsus alternans but do seem to indicate that in some instances this finding may disappear or greatly diminish when heart failure becomes advanced. It would seem that, in the presence of severe congestive failure, the ventricular filling pressure may increase to a point where it causes the diastolic stretch of
the left ventricle to be more equal from one cycle to the next. The ventricular responses would tend to become more uniform with more equal stroke outputs, and hence alternation would diminish. Support for this thesis is gained from the observations of Friedman and associates3 and our own.4 These latter studies showed that measures such as exercise, passive leg raising or infusions which increase the venous return to the heart lessen the degree of alternation. Conversely, any decrease in the venous return such as that caused by sudden tilting to the upright posture or venous tourniquets will exaggerate pulsus alternans.

We believe our findings do not conflict with previous observations that pulsus alternans may disappear or diminish with the addition of digitalis.2, 4 Such cases do not, as a rule, appear to have a severe degree of heart failure and thus digitalis may improve myocardial efficiency to the extent that alternans diminishes or disappears when cardiac function approaches normal.

All of these observations suggest that, in some instances, there may be a certain range within which the ventricular filling pressure must lie, in order for ventricular alternation to occur. Should the pressure be brought below this range, as by digitalis, alternation would disappear. On the other hand if the pressure increased beyond this range, as in severe congestive failure, alternation would also disappear. We thus would have a situation where both improvement and deterioration in cardiac function can abolish pulsus alternans.

These studies in no way lessen the significance of pulsus alternans. We, like others, have not observed this phenomenon in the absence of organic heart disease. These observations do suggest, however, that the disappearance of pulsus alternans in severe congestive heart failure may be of clinical importance in that it may be another indication that failure is progressing. They further indicate that this phenomenon might be more readily detected in certain patients after they respond to treatment for congestive failure.

**Summary**

Three patients have been observed with pulsus alternans which virtually disappeared or greatly diminished with advanced congestive heart failure.

It is suggested that this finding may be brought about by a ventricular filling pressure which has increased to the point where it causes the diastolic stretch of the left ventricle to become more uniform from one beat to the next, thus bringing about more uniform systolic ejections.

**Summario in Interlingua**

Es presentate datos in supporto del concepcion che in certe casos pulso alternante pot esser abolite o reducite in le presentia de avan-

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**Table 1.** —

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Diagnosis</th>
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<td>12m</td>
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<td>14m</td>
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tiate dysfunctionamento cardiac congestive. Le observationes pare indicar que in casos sever de dysfunctionamento congestive, le pression de replenamento ventricular pote augmentar se usque al puncto ubi illo deveni capace a render le phase diastolic del ventriculo sinistre plus uniforme ab un pulso al altere. Isto, de su parte, causarea plus uniforme ejectiones systolic con le resultato que le alternation ventricular dispares o deveni minus marcate.

REFERENCES

The Influence of Advanced Congestive Heart Failure on Pulsus Alternans
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