The Ultra Low Frequency Force Ballistocardiograph in Acute Cardiomyopathy

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SUMMARY
In this study, ultra low frequency force ballistocardiograms were recorded throughout the course of various types of acute cardiomyopathy. Conditions studied included rheumatic carditis, lupus myocarditis, sarcoid carditis, viral myocarditis, acute glomerulonephritis, idiopathic myocarditis, and familial fibrous disease of the myocardium. The instrument used was characterized by an unusually light platform and a very high performance accelerometer. In 14 of the 15 cases studied, tracings were abnormal initially; the recorded force pattern subsequently manifested progressive change which appeared to parallel the clinical course of the disease. Changes included appearance of abnormal forces in early ventricular systole, progressive change in amplitude of acceleration and deceleration forces, appearance of abnormal high frequency components in various portions of the complex, and appearance of abnormal footward forces in late systole.

The alterations thus recorded appear to offer a useful means of diagnosing and following the course of such disease entities. In some cases, this type of recording appears to provide information not available through any other conventional means.

Additional Indexing Words:
Rheumatic carditis
Viral myocarditis
Idiopathic myocarditis
Lupus myocarditis
Sarcoid carditis
Acute glomerulonephritis

ACUTE INFLAMMATION or degeneration of the myocardium may defy clinical diagnosis. There seems little doubt that myocarditis is a relatively common entity; careful postmortem studies reveal a surprising incidence of myocarditis caused by viral or bacterial infections, collagen diseases, granulomatous inflammation, and systemic toxic processes. Abelmann observed that the clinical diagnosis of myocarditis is rare, while the postmortem diagnosis is common: he concluded that the disease must often be clinically “silent.” The clinician must often base the diagnosis of myocarditis on an educated guess; objective findings tend to be sparse.

In the search for a source of objective diagnostic information in this field, the authors turned to the light-platform ultra low frequency force ballistocardiogram (ULF BCG).

The myocardium is the prime initial generator of the mechanical forces of the heart cycle. The contraction of the myocardium itself generates certain forces: more important, these forces are applied to the bolus of blood within each ventricle, generating very powerful acceleration and deceleration forces in the body. Logic suggests that any significant acute inflammation or degeneration of the heart muscle would be likely to change the magnitude and character of the forces so generated: it further suggests that detection of such abnormality of force pattern might be useful in diagnosing myocardial disease.
The authors organized this study in an attempt to answer two questions:

1. Are abnormal force patterns in fact detectable in the presence of acute inflammation or degeneration of the myocardium?

2. Is progressive change in the myocardium during the course of some acute cardiomyopathy accompanied by progressive change in the character of the force pattern of the heart cycle?

The authors have been unable to find studies of acute cardiomyopathy using this type of equipment. Earlier investigators, using the Dock shin-block apparatus or some of its variants, studied acute cardiomyopathy in rheumatic carditis, lupus erythematous, infectious diseases of various types, acute glomerulonephritis, infectious mononucleosis, trichinosis, and acute atopic reactions. These workers concluded that the BCG pattern was often deranged in the presence of acute, diffuse cardiomyopathy, with progressive alteration during the course of the disease. They concluded further that this alteration of the BCG pattern gave information not provided by any other means. These views were challenged by Abrams and Chesley who studied cases of rheumatic carditis and found no significant alteration in BCG pattern. All these studies, it must be noted, were carried out with the Dock shin-block apparatus, or the Arweit modification of this instrument. Since the ULF BCG in effect "suspends the patient in midair," it is free of certain distortions inherent in any system rigidly coupled to earth. Further, the very high-performance accelerometer used in the studies reported herein records in a frequency range not hitherto attainable. Thus the force patterns illustrated herein differ substantially, in both mechanical and electronic terms, from those described in earlier studies.

Detailed descriptions of the theory and construction of the ultra low frequency force BCG have been adequately covered in several excellent reviews. For those physicians not familiar with this field, it is worth while to point out that the ULF BCG is an extremely sophisticated instrument, free of much of the artifact which marred older BCG methods. The pendulum mounting of the bed substantially "uncouples" the patient from the earth, thereby eliminating a large source of artifact. The extremely light platform (slightly over 6 pounds) removes another source of artifact and damping. Modern high fidelity accelerometers record in excess of 80 cps, thus giving true flat response far above any of the frequencies encountered in this kind of study.

Figure 1 illustrates the correlation of the major deflections of the ULF BCG with the corresponding events of the heart cycle. Figure 2 illustrates the sharp definition and precise reproducibility of the complexes recorded with this type of instrument. We emphasize the latter point since it is crucial in this study.

A number of patients with acute cardiomyopathy were followed through the disease, and when possible, through convalescence and recovery. Each showed unmistakable clinical and laboratory signs of cardiomyopathy associated with viral infections, collagen

![Figure 1](http://circ.ahajournals.org/)

Figure 1

Correlation of major deflections of ULF BCG (acceleration) with major events of the cardiac cycle.
disease, glomerulonephritis, rheumatic fever, and in one patient, with Boeck's sarcoïd. Early in the study it became clear that progressive change in the force pattern was extremely significant. To evaluate such changes, it was necessary to know what variation in ULF BCG might be expected in a normal individual of the same age, weight, and body build during the same period of time. For each patient in this study, a control subject that is, a normal, healthy individual of the same age, weight, and body build was followed over the same period of time.

Method

The light platform used in these studies is a magnesium tubing frame across which very light nylon duck is tightly laced. Two aluminum footboards are welded to the frame. Total weight of bed with accelerometer attached is 6% pounds. Limp nylon cords suspend the bed from an overhead spring system which minimizes extraneous vibration. The sensing device is a Donner accelerometer with a flat response to 80 cps. Mechanical calibration is checked by means of a pendulum as described by Moss.27 Natural frequency of the system is 0.33 cps. All recordings were taken on a Sanborn 2 channel direct writing machine with attenuation and sensitivity settings constant for each individual. Absolute force calculations were not employed. The writers do not consider that this type of measurement is yet practical or significant in clinical terms because of extreme differences in tissue compliance from subject to subject. Each case served as its own reference for standardization.

Tracings were taken through two cycles of normal breathing, and in suspended respiration at inspiration, mid-breath, and expiration. Ample time was allowed for the subject to become relaxed and familiar with the procedure. Great care was taken to avoid a Valsalva effect. All subjects were either fasting or were at least 2 hours post cibum.

Results

All cases except one showed clear-cut abnormality of BCG pattern with progressive change which roughly paralleled the clinical course of the disease. In general, abnormal forces tended to wax as the disease reached its peak, and to wane as the pathological process in the myocardium appeared to subside.

In the "control series" minimal changes only were noted in the tracings. There were no basic changes in pattern, and there were no abnormal forces of the type recorded in...
the presence of disease. Slight widening or slurring of the I wave and changes in the H-I-J amplitude of minor degree were the maximum changes noted in the various types of cardiomyopathy.

In general, the types of abnormality demonstrated (table 1) were (1) early footward force at termination of H, 0.03 to 0.04 sec post "R" (seven cases); (2) headward force distorting the I wave, 0.08 to 0.11 sec post R (11 cases); (3) diminution of amplitude of the J (seven cases); and (4) abnormally deep K wave (five cases).

No specific pattern could be identified for any of the diseases studied. This was expected because derangement of force pattern would logically correlate with severity of myocardial pathology rather than cause.

Seven patients showed only one of the abnormalities listed above, eight showed two, and two showed three.

Figures 3 and 4 illustrate two of the cases studied together with their normal controls. In the case of the sarcoid heart disease (fig. 3), a very powerful abnormal headward force appears in early systole in the tracing of January 14, 1964. This force is still present, although very greatly diminished in the tracing of February 4, 1964, and has completely disappeared in the tracing of March 21, 1964. The same phenomenon is illustrated in the case of acute glomerulonephritis (fig. 4) with a powerful abnormal headward force in the tracing of August 16, 1963, which has completely disappeared in the tracing of October 9, 1963. No change whatever took

Figure 3
Sarcoid heart disease with cardiomegaly, murmur, abnormal ECG, and positive biopsy of pulmonary lesion in a female, aged 16 years. Patient was acutely ill at time of first tracing (A); dramatic improvement from corticosteroid therapy had occurred at time of second tracing (B); patient was asymptomatic and ambulatory at time of the third tracing (C). Note that the abnormal early headward force in A was diminished in B, and absent in C. (Control tracings A to C) Subject was a healthy female, of same age, weight, and body build as the patient; same interval between tracings.
# Types of Abnormality Demonstrated

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>H</th>
<th>I</th>
<th>J</th>
<th>K</th>
<th>General</th>
</tr>
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<tbody>
<tr>
<td>Rheumatic carditis</td>
<td>Early</td>
<td>Late</td>
<td>Early</td>
<td>Late</td>
<td>Diminished amplitude</td>
</tr>
<tr>
<td>A.B.</td>
<td>0.03 sec†</td>
<td>0.06 sec</td>
<td>Dim.†</td>
<td></td>
<td>Changing P-R interval</td>
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<tr>
<td>L.B.</td>
<td>0.04 sec†</td>
<td></td>
<td></td>
<td></td>
<td>Bizarre deformity</td>
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<tr>
<td>J.C.</td>
<td>0.04 sec†</td>
<td>0.10 sec†</td>
<td>Dim.†</td>
<td></td>
<td>changing P-R interval</td>
</tr>
<tr>
<td>C.M.</td>
<td>0.04 sec†</td>
<td></td>
<td></td>
<td></td>
<td>Notched I-J segment</td>
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<tr>
<td>R.P.</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>W.T.</td>
<td>0.06 sec†</td>
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<tr>
<td>R.C.</td>
<td></td>
<td>0.11 sec†</td>
<td>Dim.†</td>
<td></td>
<td>Deep</td>
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<tr>
<td>S.B.</td>
<td></td>
<td>0.075 sec†</td>
<td></td>
<td>Dim.†</td>
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<td>S.M.</td>
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<td></td>
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<td>Dim.†</td>
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<tr>
<td>D.W.</td>
<td></td>
<td></td>
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<td>Dim.†</td>
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<tr>
<td>P.S.</td>
<td></td>
<td></td>
<td></td>
<td>Dim.†</td>
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<td>Systemic lupus erythematous</td>
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<td>M.M.</td>
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<tr>
<td>S.S.</td>
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<tr>
<td>J.M.</td>
<td></td>
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<tr>
<td>Acute glomerulonephritis</td>
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<td>J.B.</td>
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<tr>
<td>W.Q.</td>
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<tr>
<td>Viral myocarditis</td>
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<td>N.K.</td>
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<td>H.B.</td>
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<td>C.M.</td>
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Circulation, Volume XXXVI, July 1967

PHIBBS ET AL.
Acute hemorrhagic glomerulonephritis with azotemia in a man aged 42 years: (A) Abnormal ECG, tachycardia, and apical murmur; (B) Patient was ambulatory after coil dialysis and convalescence: glomerulonephritis had abated and was asymptomatic; urine was negative. Note prominent early headward force in first tracing (A) and its absence in the second (B). (Control tracings) Healthy male of same weight and body build; same interval between tracings.

Figures 5 to 7 illustrate three characteristic progressions of ULF BCG patterns during the course of acute cardiomyopathy. In the case of lupus erythematosus (fig. 5), the striking deformity in the tracing of March 19, 1963, needs little description; footward acceleration is greatly diminished and is interrupted by an abnormal headward force. A deep K wave is present. In the tracing taken when the patient was clinically greatly improved on April 11, 1963, the BCG is substantially normal. In the case of acute rheumatic fever (fig. 6), the progression from a relatively normal ULF BCG at onset of disease to an almost unrecognizable, bizarre set of deflections on November 3, 1962, is particularly striking. This patient had an extraordinarily severe, fulminating type of rheumatic fever: even the symptoms of polyarthritis were difficult to control with corticosteroids. The carditis was very severe with tachycardia, atrioventricular (A-V) block, loud murmur, x-ray evidence of enlargement, and a short period of congestive heart failure.

The patient with viral myocarditis (fig. 7), similarly manifested alarming evidence of cardiac pathology with acute enlargement of the heart, tachycardia, and congestive heart failure early in the course of the disease. The appearance of an abnormal headward force deforming the wave of footward acceleration is clearly illustrated in the tracing of April 8, 1962. This force is still detectable in tracings even as late as October 14, 1962, when the patient was ambulatory. At this time it is greatly diminished, and appears as a small notch on the H-I downstroke.

Discussion

The results of this study bear out the initial hypothesis that pathological change in the myocardium will alter the force pattern of the heart cycle. How are these changes produced? Imagine the force imparted to a bolus of fluid if one squeezed a bulb syringe with an even, rapid, forceful movement. Now imagine the hand doing the squeezing to be severely inflamed with painful cellulitis: The expulsion of fluid will be slower, and the force will tend to be irregular and unevenly applied. Much the same process can be assumed in acutely inflamed or degenerated myocardium. The acceleration imparted to the mass of blood by ventricular contraction will be erratically applied, with varying degrees of force at various points around the
Figure 5

Patient critical with severe carditis at time of tracing B. Excellent remission with corticosteroid treatment at time of tracing C.

Figure 6

Very severe rheumatic carditis. Cardiomegaly, murmur, and increasing P-R interval at time of tracing B. At time of tracing C carditis and polyarthritis were still severe. At time of tracing D murmur was loud; clinical remission was beginning. Corticosteroid treatment was begun. At time of tracing E patient was clinically improved; carditis apparently was subsiding.

Figure 7

(A) Patient was acutely ill. (B) Clinical improvement with digitalis and diuretic therapy. (C) Patient was ambulatory; functional ability was grade 3. (D) Patient was employed; functional ability was grade 2.
ventricles. The time sequence of acceleration and deceleration forces will be altered, and the magnitude of these forces would naturally change.

Is the ULF BCG more informative than other diagnostic means, for example, the ECG? Our series is not large enough to permit conclusions. In three cases, however, with serious proved cardiomyopathy, the ECG was normal, while the ULF BCG was unmistakably abnormal. One was a case of lupus erythematosus in which postmortem study revealed massive, diffuse angitis of the myocardial vascular bed. An ECG recorded 16 days before death was normal. The BCG showed obvious gross deformity of footward acceleration. The second was the case of sarcoidosis illustrated in figure 3 with confirmatory pulmonary biopsy. The ECG was normal except for a period of ventricular bigeminy. In the third case, disseminated lupus erythematosus, ECG change was limited to transient P-R prolongation. Changing murmur, tachycardia, and dyspnea were among the manifestations of carditis. The grossly abnormal BCGs are illustrated in figure 5.

Although a different instrument was employed, this study tends to confirm the observations of Dolan, Mendelbaum and Arbeit and his associates cited earlier. We think that the negative observations of Abrams and Chesley may have been due to technical problems. Certainly much of the change recorded in our series was in the higher frequency ranges and would not have been detected with a less responsive system.

Conclusions

It is concluded that acute diffuse cardiomyopathy often produces progressive change in the pattern of contractile force of the myocardium, which change is mirrored in the ULF BCG. The alterations thus recorded appear to offer a useful means of diagnosing and following the course of such disease entities. In some cases, this type of recording appears to provide information not available through any other conventional means.

References


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Tractatus de Corde, 1669

Right-to-Left Shunt Through Foramen Ovale Illustrated.

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Circulation, Volume XXXVI, July 1967
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Circulation. 1967;36:92-100
doi: 10.1161/01.CIR.36.1.92
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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