Ballistocardiographic Study in Severe Anemia

By O. P. Tandon, M.D., and B. C. Katiyar, M.D.

Anemia affects the heart by impairing the oxygen supply of the myocardium and by increasing the mechanical load on the heart through alterations in the circulatory dynamics. Irrespective of the cause of anemia, the cardiac and circulatory disturbances are identical as long as the anemia is severe. With the introduction of ballistocardiographic techniques, a new tool became available for the study of the force of cardiac contractions in various disease states; this technic, however, has not been adequately exploited to investigate the cardiac changes in anemia. Dock and his colleagues made only casual references to the ballistocardiographic changes in anemia, stating that the tracings showed high-amplitude waves indicating increased force of cardiac contraction. They noted that the correction of anemia caused a reversion of the high-amplitude waves toward normal. In a study of severely anemic patients, Sanghvi and Banerjee noted abnormal tracings in every case, the abnormalities consisting of tall or bifid H; fused HJ; broad or bifid J; short, absent, or notched K; tall L, M, or N; and totally bizarre complexes of low amplitude. In a third of their cases, large systolic complexes were seen, and the pattern was grossly abnormal in few of these. In significant contrast to the high-amplitude waves, totally bizarre complexes of small amplitude and grade-IV abnormality were seen in the minority of cases. These authors noted that, in the vast majority of cases, ballistocardiographic abnormalities persisted even after the cure of anemia and that the degree of abnormality often remained unaltered by the correction of anemia. In order further to document the ballistocardiographic changes, a study of 10 cases of severe anemia is reported.

Material and Methods

The present study consists of 10 cases of severe anemia due to hookworm infestation. The age, sex, weight, and hemoglobin levels (before and after the treatment) are given in table 1.

The presence of any clinically detectable heart disease was excluded. The study was carried out by Dock's direct body ballistocardiograph with the electromagnetic pick-up. Hospital beds being unstable and therefore unsuitied for ballistocardiographic study, a rigid hard wooden board was used during the recording of the ballistocardiogram. A double-channel electrocardiographic machine was used to record the tracings. On channel A of the machine, the ballistocardiogram was taken while channel B was used to record the lead II of the electrocardiogram. The tracings were recorded in the recumbent position with the breath held in normal respiration, in deep inspiration (with the mouth open to avoid Valsalva effect), and again in deep expiration. The instrument was standardized so that a deflection of 10 mm. was obtained with a current of 1 mv. In each case, the ballistocardiographic pattern was studied before and after the treatment.

As a control, 20 healthy normal subjects were studied. The tracings of these normal individuals (fig. 1) were used to determine the maximum normal range of height and duration of individual waves. The waves outside this range were regarded as abnormal. On the basis of this control series the various waves have, in subsequent results, been designated as small, tall, deep, or wide. The criteria for these designations are given in table 2. The records were graded as I, II, III, and IV based on the criteria of Brown et al.3 4

Results

All the tracings on admission showed ballistocardiographic abnormalities (table 3 and figs. 2-6). Detailed changes are sufficiently evident by inspection of the tables and figures and therefore are not described here.

The ballistocardiographic abnormalities can be summarized as follows: 1. Before treatment, most of the waves were smaller than normal. They showed a tendency to regain their normal size after treatment. 2. In few tracings, the waves were taller than normal after treatment (figs. 3 and 4) but never before treatment (except in 2 cases where J wave was tall even on admission). 3. There were tracings where waves remained smaller in size even after treatment. It was possible
Figure 1

Ni and Nl are tracings from 2 clinically normal subjects.

Table 1

Clinical Data in Ten Cases of Anemia

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age</th>
<th>Sex</th>
<th>Weight in pounds</th>
<th>Hemoglobin in grams per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Before treatment</td>
<td>After treatment</td>
</tr>
<tr>
<td>1</td>
<td>50</td>
<td>M</td>
<td>130</td>
<td>4.0</td>
</tr>
<tr>
<td>2</td>
<td>30</td>
<td>M</td>
<td>122</td>
<td>2.6</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>F</td>
<td>105</td>
<td>5.2</td>
</tr>
<tr>
<td>4</td>
<td>25</td>
<td>M</td>
<td>110</td>
<td>3.5</td>
</tr>
<tr>
<td>5</td>
<td>28</td>
<td>M</td>
<td>118</td>
<td>3.0</td>
</tr>
<tr>
<td>6</td>
<td>30</td>
<td>F</td>
<td>98</td>
<td>5.8</td>
</tr>
<tr>
<td>7</td>
<td>35</td>
<td>F</td>
<td>108</td>
<td>3.0</td>
</tr>
<tr>
<td>8</td>
<td>28</td>
<td>F</td>
<td>114</td>
<td>3.0</td>
</tr>
<tr>
<td>9</td>
<td>24</td>
<td>M</td>
<td>104</td>
<td>4.0</td>
</tr>
<tr>
<td>10</td>
<td>19</td>
<td>F</td>
<td>85</td>
<td>6.0</td>
</tr>
</tbody>
</table>

that they might have attained their normal size by the time the hemoglobin had reached normal. Actually all cases were discharged before normal hemoglobin could be attained. 4. Only 1 case showed grade-IV changes, and by treatment it reverted back to grade I (fig. 2). 5. No case showed fused HJ complexes, M patterns, or high-amplitude curves.

Discussion

On admission, all 10 cases showed abnormal ballistocardiograms. Of these 10 cases, 8 showed ballistocardiographic abnormalities even after improvement of anemia, either due to persistence of the previous abnormalities or due to appearance of newer ones during the course of treatment. The abnormalities consisted of small amplitude curves and nonspecific changes in the waves.

Small Amplitude Curves

On admission, most of the ballistocardiograms showed low-amplitude waves. Such low-amplitude curves, although found in the minority of cases, have also been reported by Sanghvi et al.² In physiologic terms, these low-amplitude curves indicate decreased force of cardiac contraction. It has been postulated that the state of chronic severe anemia causes the anoxic myocardial damage that is responsible for the cardiac weakness. On improvement of anemia, curves showed a tendency to revert to normal, owing to elimination of the anoxic factor. Certain tracings were not completely restored to normal, probably due to persistence of anemia as a result of inadequate treatment.

High-amplitude curves in anemia, although not seen in any of our cases, have been re-
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Figure 3
NA (a) and (b) are tracings taken before and after treatment, respectively. Before treatment, II and M waves were small. The other waves were in normal range. After treatment, the complexes showed high-amplitude curves.

Figure 4
J (a) and (b) are tracings taken before and after treatment, respectively. Prior to treatment, H and M waves were small and L wave was wide. After treatment, all waves show high amplitude.

Table 3
Ballistocardiographic Observations in Ten Patients with Anemia

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Name</th>
<th>Waves (measured in mm.)</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>G</td>
<td>S N N N N N S S S</td>
<td>Before treatment</td>
</tr>
<tr>
<td>2.</td>
<td>Na</td>
<td>S N N N N — S N N</td>
<td>Before treatment</td>
</tr>
<tr>
<td>3.</td>
<td>K</td>
<td>S N N N S T T S</td>
<td>Before treatment</td>
</tr>
<tr>
<td>4.</td>
<td>VP</td>
<td>N N T N N S S S S S</td>
<td>Before treatment</td>
</tr>
<tr>
<td>5.</td>
<td>M</td>
<td>S N N N S S S S S S S</td>
<td>Before treatment</td>
</tr>
<tr>
<td>7.</td>
<td>RP*</td>
<td>N N N N N N W S — — —</td>
<td>Before treatment</td>
</tr>
<tr>
<td>8.</td>
<td>KK</td>
<td>N N N N — — — — — — —</td>
<td>Before treatment</td>
</tr>
<tr>
<td>9.</td>
<td>MM</td>
<td>S N N N N — — — — — —</td>
<td>Before treatment</td>
</tr>
</tbody>
</table>

*Before treatment all complexes show grade IV changes.
—, Absent; N, normal; W, wide; T, tall; S, small.
portated by many workers.\textsuperscript{1, 2} Besides anemia, large ballistic complexes are also seen in many other cardiovascular diseases of high-output type, like arteriovenous fistulas, thyrotoxicosis, emaciation, patent ductus arteriosus, and certain pulmonary conditions.\textsuperscript{5-9} The high-amplitude patterns in these conditions tend to revert to normal when treated. High-amplitude curves in anemia are due to decreased peripheral resistance against which the left ventricle must work. Absence of such curves in the present series was probably due to the more chronic severe nature of anemia, so that the anoxic left ventricle was weakened.

**Individual Wave Abnormalities**

Various ballistic waves showed nonspecific changes. Prior to treatment, the abnormalities consisted of small H waves; small, tall, wide, or normal J waves; normal or small K waves; and normal, absent, or small LM and N waves. Only 1 case showed grade-IV abnormalities.

In the present series, the H wave was small in 7 cases. De Lalla et al.\textsuperscript{10} have analyzed the genesis of the H wave and have suggested that both atrial contraction and the thrust of the heart during isometric contraction are involved in the origin of the H wave. As in chronic severe anemia, both the atrial and ventricular contractions are weakened, due to anoxic myocardial damage, resulting in small H waves. In contrast to findings of Sanghvi et al., no tracings in our series showed tall H waves on admission. These workers believed that tall H waves in their cases were due to increased upward flow of blood in the atria during isometric contraction as a result of anoxic myocardial damage.

The K wave was found to be normal in most of the tracings of our series. Only 2 cases showed small K waves, and these became normal after treatment. It is difficult to decide whether small K waves in these 2 cases were due to decreased peripheral resistance or due to weakened myocardium. Sanghvi et al.\textsuperscript{2} found small K waves in most of their cases. These workers have attributed these small K waves to decreased peripheral resistance in anemic patients. Short K waves have also been noted in a variety of other conditions like aortic stenosis,\textsuperscript{4} hypotension,\textsuperscript{7, 11} coarctation of the aorta,\textsuperscript{12} vertical hearts, and thrombosis of the abdominal aorta.\textsuperscript{13}

In contrast to findings of Sanghvi et al.,\textsuperscript{2} none of our cases showed fused HJ complexes, notched I, J, or JK segments, or notched J waves.

On improvement of anemia, a few fresh abnormalities were seen. These consisted of tall H waves (1 case), tall I waves (1 case), tall J waves (3 cases), and tall (3 cases) and wide (2 cases) L waves. In older subjects, known to have heart trouble, Dock et al.\textsuperscript{1} also noted the appearance of abnormal ballistic patterns on improvement of anemia. These workers found that only patients without heart disease showed a reversal of high-amplitude curves toward normal without appearance of any fresh abnormality.

**Summary**

Ballistocardiographic study in 10 cases of chronic severe anemia, with use of Dock's direct body ballistocardiograph, has been reported. The tracings were studied before and after treatment of anemia.

All cases showed abnormal ballistocardiograms. The abnormalities presented no specific pattern. Prior to treatment, in the majority of cases, the waves were smaller than normal.
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With few exceptions, the tracings tended to regain their normal size on correction of anemia. No tracings showed fused HJ complexes, tall H waves, notched J waves, M patterns, or high-amplitude curves either before or after treatment.

The present study shows that the state of severe anemia of chronic duration, impairs the mechanical force of cardiac contractions, and, on improvement of anemia, the functional state of the heart improves. Thus, it reflects the usefulness of this new tool in assessing the functional status of the heart in patients with chronically severe anemia.

References

This velocity (systolic) is only the velocity of the blood at its first entering into the aorta, in the time of systole; in consequence of which the blood in the arteries, being forcibly propelled forward, with an accelerated impetus, thereby dilates the canal of the arteries, which begin again to contract at the instant the systole ceases; by which curious artifice of nature the blood is carried on in the finer capillaries, with an almost even tenor of velocity, in the same manner as the spouting water of some fire-engines is contrived to flow with a more even velocity, notwithstanding the alternate systoles and diastoles of the rising and falling embolus or force.—Stephen Hales, B.D., F.R.S. Haemastatics, Vol. II, London, 1733.
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