Calibrated Low Frequency Acceleration Vibrocardiography
Its Hemodynamic Determinants and Clinical Application

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Summary
Calibrated low frequency acceleration vibrocardiography (A-VCG) was studied in normal individuals and in patients with coronary heart disease. Experimental studies in anesthetized dogs were also made to look for the hemodynamic determinants of the main waves in the A-VCG, and transmission characteristics of the chest wall over the range of the frequency of the A-VCG. The results were as follows:

1. The S wave of low frequency acceleration vibrocardiography (A-VCG) coincided with the upward slope of the aortic flow rate curve, but showed a reverse phase, and its trough coincided with the vertex of the first derivative of aortic flow rate.

2. The amplitude of the S wave was correlated with the peak value of aortic flow rate, the peak value of the first derivative of aortic flow rate and the peak value of the first derivative of aortic pressure, but was not correlated with aortic pressure, stroke volume and the peak value of the first derivative of left ventricular pressure.

3. In the patients with myocardial infarction, there was a significant increase in A wave amplitude, decrease in S wave amplitude and increase in A/S ratio compared with those of normal individuals.

4. In the patients with angina pectoris, there was a significant increase in A/S ratio compared with that of normal individuals with the age of between 30 and 39.

5. In normal individuals, there was tendency for A wave amplitude to increase, S wave amplitude to decrease and A/S ratio to increase with age.

6. During exercise and inhalation of amyl nitrite, increase in S wave amplitude was smaller in the patients with angina pectoris than in normals.

7. These results suggest that the S wave amplitude in A-VCG reflects contractility of the left ventricle, and that the A/S ratio and changes in S wave amplitude during various conditions offer useful means for detecting latent impairment of the functional reserve of left ventricle.

8. The wave form and phase of direct A-VCG from the ventricular wall and those from the chest wall were similar, and the S wave amplitude was about 6 dB greater in direct A-VCG.

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The outline of this study was reported at the 33rd Annual Meeting of the Japanese Circulation Society in 1969 (Tokyo) and at the 34th Annual Meeting of the Japanese Circulation Society in 1970 (Kobe).
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INFORMATION about hemodynamics is indispensable for clinical management of cardiovascular patients and for evaluating their prognosis. Many indices of the hemodynamics have been proposed, but most of them include surgical methods and are often impractical for routine clinical application, even if they are so useful. A simple, non-surgical and indirect method has been desired.

The first attempt to evaluate hemodynamics through vibration produced by cardiac activity and blood flow was made by Goldon in 1877. Later, Starr described fundamental studies and clinical application of a ballistocardiography. Although a ballistocardiography has been applied for an evaluation of coronary heart disease, it seems to be too complicated for a routine examination because of its poor reproducibility and of difficulty in keeping the platform constant. Furthermore, the evaluation tends to vary depending upon judgement of the examiner.

On the other hand, an attempt was made to record the vibration accompanying cardiac movement and blood flow through the chest wall and was reported by Marey and Potain in 1885. They attempted to record displacement of chest wall by optical means, however, the criteria of abnormal wave form were not so highly reliable as in cases of ballistocardiography.

There have been many reports on recording the displacement of chest wall vibration thereafter, such as Luisada and others’ low frequency vibrocardiography, Eddleman and others’ kinetocardiography, Skinner’s kinetocardiography, Benchimol’s apexcardiography, Ueda and others’ precordial low frequency vibrocardiography. Agress and others devised a vibrocardiography that records the velocity of chest wall vibration.

Acceleration vibrography for recording acceleration of chest wall vibration was pursued by Landes, Rosa, Mounsay, and Hollis respectively. These devices are said to be simpler way in recording, have better reproducibility, and be easier for evaluation, compared with displacement vibrocardiography.

The drawback which is common with these methods is the difficulty in the calibration of amplitude, therefore no attempt has been so far made for quantitative measurements.

Machii and others developed a compact and light-weight precordial accelerometer, calibrated by the gravitational acceleration, and tried quantitative measurement on the main waves of precordial vibration in order to evaluate...
I, the author, hereby wish to deal with the low frequency acceleration vibrocardiography (A-VCG) with the use of afore-said accelerometer and follow experimentally the correlation between the amplitude of main waves and some indices of hemodynamics, and finally discuss the possibility of clinical application of this method by comparing normal values of A-VCG with those from the patients with coronary heart disease.

**Materials and Methods**

1) Experiment
   i) Animal experiment

   Fifteen mongrel dogs (body weight 11.0–17.0 Kg., average of 13.7 Kg.) were used. After intravenous anesthesia by Pentobarbital (Nembutal®, 25 mg./Kg.), the animal was fixed in supine position, median sternotomy was performed, 3 catheters were simultaneously inserted into the left atrium, aorta and left ventricle to measure pressure, and a probe of electromagnetic flow-meter was set at the aortic root. After the incision was closed, a transducer was fixed on the chest wall at the site of apical pulse (Fig. 1). With the certain 4 dogs, another transducer wrapped in a thin latex film was sewed, in addition, directly on the left ventricular wall (Fig. 2).

   Left ventricular pressure (P_LV) and its first derivative (dP_LV/dt), aortic pressure (P_AO) and its first derivative (dP_AO/dt), and aortic flow rate (F) and its first derivative (dF/dt), were also recorded simultaneously with A-VCG. Several substances, such as Isoproterenol, Propranolol, amyl nitrite, Lanatoside C, Prostirallidin A and Noradrenaline, were administered to observe hemodynamic alterations by means of these parameters.

   The values represent average figures with each of the successive 3 waves.

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![Fig. 1. The transducer is attached to the chest wall. The dog is fixed in a supine position.](image)
Fig. 2. The transducer is attached directly to the ventricular wall. Electromagnetic flowmeter is seen at the root of aorta. (dog)

ii) Experiment on Human Chest Wall
A-VCG was recorded, with regard to 2 normal male individuals, at 7 sites respectively on the chest wall, and comparative examinations of the amplitude and the phase of S wave were made on each recording. The sites studied were (1) 5th intercostal space on the midclavicular line, (2) 4th intercostal space on the left sternal border, (3) 3rd intercostal space on the left sternal border, (4) 2nd intercostal space on the left sternal border, (5) 2nd intercostal space on the right sternal border, (6) 4th intercostal space level on sternum and (7) 4th intercostal space on midclavicular line.

At the same time, A-VCG were recorded on a tape with a data recorder, and the frequency analysis was carried out by means of a 1/3 Oct. band pass filter and a high-speed level recorder.

iii) Chest Wall Transmission in Anesthetized Dogs
From one transducer directly attached to the ventricular wall and another transducer on the chest wall, A-VCG were simultaneously recorded with a photographic recorder and an analogue data recorder. The frequency of these records were analysed by means of 1/3 Oct. band pass filter and a high-speed level recorder.

2) Clinical Application
A-VCG was recorded from the transducer fixed with an adhesive tape at the site of apex on the chest wall, or at the site of 5th intercostal space on the midclavicular line when the apical pulse was indistinct (Fig. 3). The values of the waves of A-VCG were represented by the average amplitudes of waves in each of 3 successive cardiac cycles.

i) Normal Individuals
A total of 35 cases, with normal blood pressure, normal X-ray findings, normal ECG findings and without signs and symptoms suggestive of heart disease were selected for normal controls. They were divided into 4 different groups of (1) 7 cases, aged from 10 to 19, (2) 7 cases, 20 to 29, (3) 13 cases, 30 to 39, (4) 8 cases, 40 to 49. Cases above the age of 50 were excluded for fear of existing latent coronary heart disease.
ii) The Patient with Angina Pectoris
A total of 25 cases having a past history of anginal attacks and with abnormal ECG findings were subjected to recording. Their age ranged from 30 to 50.

iii) The Patients with Myocardial Infarction
A total of 18 cases, aged 40 to 67, diagnosed by clinical symptoms, ECG and blood chemistry were subjected to recording. The occurrence of the latest attacks dated back from 1 week to 2 years ago.

iv) Amyl Nitrite Inhalation Test
Amyl nitrite was inhaled for 10 sec. by 10 normal cases and 6 cases with coronary heart disease, and their A-VCG was taken once before inhalation, after which it was conducted 15 times altogether with intervals of 10 sec.

v) Exercise Test
The Master's two-step test (single) was administrated to 8 normal cases and to 6 cases with coronary heart disease, and an A-VCG was recorded once before exercise, after that, the recording was made 3 times at 3, 5, and 7 min. after exercise.

APPARATUS

Transducer: A low frequency accelerometer; Model PM-01, made by Rion Co., Ltd.; Weight 40 Gm.; Diameter 2.0 cm.; Element: lead zirconate-barium titanate, dumped by silicone oil. Sensitivity: 1.1 V/G (G: the gravitational acceleration). Frequency response: linear from 3 to 700 Hz. Calibrated by 22 mV. (10 Hz) as $2 \times 10^{-2}$ G (G: the gravitational acceleration) (Fig. 4).

Amplifier: Specially made by Rion Co., Ltd., with linear frequency response from 2 to 10^2 Hz.

Recorder: Photographic oscillograph, made by Sanei Sokki Co., Ltd.; Model 100 A, linear from DC to 300 Hz. Paper speed, 100 mm./sec.

Aortic Flow Rate: Measured by an electromagnetic flow meter; Model MF-5, by Nihon Kôden Co., Ltd.
RESULTS

1) Principal Configuration of A-VCG Waves

As shown in Fig. 5, there were A, S, and V waves in an A-VCG. The A wave was considered to be an atrial wave, and consisted of 1 or 2 peaks appearing between P and R wave in ECG and approximately coincided with the fourth heart sound in PCG. Its amplitude ranged from 0.005 G to 0.025 G.

The S wave was considered to accompany left ventricular ejection. It had biphasic deflections and its trough was, in an average, 0.16 sec. (0.14–0.17 sec.) behind the beginning of the Q wave of ECG. S wave was the largest wave in A-VCG, and the amplitude ranged from 0.01 G to 0.08 G.

The V wave was considered to appear at the time of ventricular rapid filling and approximately correspond to the third heart sound in PCG.

2) Hemodynamic Determinants and the S Wave Amplitude

The S wave corresponded to the rise of aortic flow rate (F), but showed a reverse phase and its trough corresponded to the vertex of the first derivative of aortic flow curve (dF/dt). The S wave amplitude showed a good correlation
Fig. 5. Configuration of A-VCG in a normal. 26 year old male.

Table I. Correlation Coefficient between the Amplitude of the S-Wave and Hemodynamic Determinants

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Peak F</th>
<th>Peak dF/dt</th>
<th>Peak dP_a/dt</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.84</td>
<td>0.96</td>
<td>0.85</td>
</tr>
<tr>
<td>2</td>
<td>0.86</td>
<td>0.88</td>
<td>0.77</td>
</tr>
<tr>
<td>3</td>
<td>0.65</td>
<td>0.89</td>
<td>0.76</td>
</tr>
<tr>
<td>4</td>
<td>0.75</td>
<td>0.90</td>
<td>0.76</td>
</tr>
<tr>
<td>5</td>
<td>0.75</td>
<td>0.92</td>
<td>0.76</td>
</tr>
<tr>
<td>6</td>
<td>0.67</td>
<td>0.77</td>
<td>0.82</td>
</tr>
<tr>
<td>7</td>
<td>0.67</td>
<td>0.86</td>
<td>0.65</td>
</tr>
<tr>
<td>8</td>
<td>0.82</td>
<td>0.92</td>
<td>0.94</td>
</tr>
<tr>
<td>9</td>
<td>0.65</td>
<td>0.94</td>
<td>0.72</td>
</tr>
</tbody>
</table>

with the peak flow rate (peak F), peak value of dF/dt (peak dF/dt) and the peak value of the first derivative of aortic pressure (peak dP_a/dt), as shown in Table I, and in Figs. 6 to 10. The S wave was not correlated to aortic pressure, mean aortic flow, stroke volume and the peak value of the first derivative of left ventricular pressure (peak dP_LV/dt).

3) The A-VCG of Normal Subjects and Variations thereof Due to Age

The A wave amplitude was maximum in cases from 10 to 19 years old and minimum in cases from 20 to 29, and tended to increase thereafter with age. The amplitude in cases from 10 to 19 was significantly greater (P < 0.05) than those in cases from 20 to 29, 30 to 39, and 40 to 49, respectively. There was no
Fig. 6. Configuration of A-VCG and several hemodynamic indices in an anesthetized dog. The beginning of the S wave deflection corresponds to the beginning of flow curve, and the bottom of the S wave deflection corresponds to the peak of dF/dt.

Fig. 7. Correlation between the amplitude of the S wave and the peak value of dF/dt. (the first derivative of aortic flow rate)
Fig. 8. Correlation between the amplitude of S wave and the peak aortic flow rate.

Fig. 9. Correlation between the amplitude of the S wave and the peak value of $dP_{Ao}/dt$. (the first derivative of aortic pressure.)

statistically significant difference between the amplitude in cases from 20 to 29 and that in those from 30 to 39. The amplitude in cases from 40 to 49 was significantly greater ($P<0.05$) than that in those from 20 to 29, and 30 to 39.

The S wave amplitude was maximum in cases from 10 to 19 years old and tended to decrease with age. The amplitude in those from 10 to 19 was significantly greater ($P<0.05$) than that in those from 40 to 49. The amplitude in those from 20 to 29 was significantly greater ($P<0.05$) than that in those from 40 to 49, but not significantly different from that in those from 30 to 39.

The A/S ratio was maximum in forties and minimum in twenties. There
Fig. 10. Distribution of the A wave amplitude, the S wave amplitude and the A/S ratio in normals of different age groups, angina pectoris cases and myocardial infarction cases. The amplitude is expressed by gravitational acceleration unit (G).

Table II. Amplitudes of the A and S Wave, A/S Ratio in Normals of Different Age Groups, Angina Pectoris and Myocardial Infarction Cases

<table>
<thead>
<tr>
<th>Age (yrs.) (N)</th>
<th>A wave</th>
<th>S. D.</th>
<th>S wave</th>
<th>S. D.</th>
<th>A/S ratio</th>
<th>S. D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>10–19 (7)</td>
<td>1.63</td>
<td>1.23</td>
<td>6.65</td>
<td>0.95</td>
<td>0.275</td>
<td>0.169</td>
</tr>
<tr>
<td>20–29 (7)</td>
<td>0.64</td>
<td>0.17</td>
<td>3.62</td>
<td>1.07</td>
<td>0.199</td>
<td>0.040</td>
</tr>
<tr>
<td>30–39 (13)</td>
<td>0.70</td>
<td>0.12</td>
<td>3.11</td>
<td>0.79</td>
<td>0.246</td>
<td>0.112</td>
</tr>
<tr>
<td>40–49 (7)</td>
<td>1.02</td>
<td>0.25</td>
<td>2.76</td>
<td>1.02</td>
<td>0.350</td>
<td>0.018</td>
</tr>
<tr>
<td>Angina (23)</td>
<td>0.91</td>
<td>0.43</td>
<td>3.38</td>
<td>1.41</td>
<td>0.520</td>
<td>0.382</td>
</tr>
<tr>
<td>Infarction (18)</td>
<td>1.55</td>
<td>1.02</td>
<td>2.53</td>
<td>0.22</td>
<td>0.513</td>
<td>0.202</td>
</tr>
</tbody>
</table>

(G: Gravitational acceleration)

were significant differences in A/S ratio among twenties, thirties, and forties respectively (P<0.05). The A/S ratio in teens was significantly greater (P<0.05) than that in twenties, but did not show significant difference with that in thirties and forties (Fig. 10, Table II).

4) The A-VCG in Patients with Coronary Heart Disease

As shown in Figs. 11, 14, and Table II, the A wave amplitude, the S wave amplitude and the A/S ratio in patients with angina pectoris did not show significant differences from those of normals in forties, but a significantly greater A/S ratio was seen (P<0.05) when compared with that of normals in thirties.
Patients with myocardial infarction showed significantly greater A wave amplitude, smaller S wave amplitude and increased A/S ratio when compared with those in normals in forties ($P < 0.05$).

The A wave amplitude was significantly greater in cases with myocardial infarction than that in cases with angina pectoris ($P < 0.05$), but the S wave amplitude and the A/S ratio did not show significant differences in those 2 groups.

5) A-VCGs during Exercise Test

In normal individuals, no significant difference was observed in A wave amplitude before and immediately after exercise, but the S wave amplitude showed a significant increase of an average of 81% and significant decrease in the A/S ratio of an average of 17% ($P < 0.05$) (Fig. 12).

In the angina pectoris group, there was a significant increase of an average of 40% in the A wave amplitude before and immediately after exercise ($P < 0.05$), but the increase of an average of 14% in the S wave amplitude was not significant, a significant increase was also observed in the A/S ratio ($P < 0.05$).

6) Amyl Nitrite Inhalation Test

In normal individuals, there was a significant increase of an average of 37.3% in the A wave amplitude and an average of 80.2% in S wave amplitude after lapse of 20 sec. after the beginning of inhalation ($P < 0.05$), but the A/S ratio showed no significant difference.
In the angina pectoris group, there was no significant difference in the A wave amplitude, S wave amplitude and the A/S ratio before and after inhalation (Fig. 13).

7) Changes in the Wave Form and the Amplitude of the Main Waves
Due to Different Sites of Recording of the Human Chest Wall

Around the apex, there was little difference in phase and amplitude of the S wave according to the position of recording. The phase was reversed at the
Fig. 14. Frequency distribution of A-VCG from 7 different sites of the chest wall in a normal 26-year-old male. Each number on curves shows the site of recording. (refer the text)

Fig. 15. A-VCG recorded directly from ventricular wall and chest wall; Both configurations are similar in waveform and phase.
There was little change in phase and amplitude of the A wave.

8) A-VCG in Dogs from the Ventricular Wall and from the Chest Wall

There was no great difference in frequency distribution between the recordings from the ventricular wall and from the chest wall except that, in the frequency range above 100 Hz, A-VCG components from the chest wall were lower than those directly recorded from the ventricular wall. The level of the direct A-VCG from the ventricular wall showed about 6 dB higher amplitude (Figs. 14, 15).

9) Frequency Distribution of A-VCG in Human and Anesthetized Dogs

Both showed the same tendency (Figs. 16, 17).

**DISCUSSION**

The origin of low-frequency vibration in the chest wall may be due to
vibration accompanying cardiac activity, especially due to changes in the tens-
ion of the myocardium at the time of ejection, and vibration due to pulsation of
great vessels, particularly due to pulsation produced by blood impact.

In considering main components of the A-VCG—i.e. the A, S, and V
waves,—the A wave is thought to be a vibration accompanying atrial contrac-
tion, because it coincided with the fourth heart sound and disappeared in atrial
fibrillation, but was observed with the P wave of the ECG in complete A-V
block (Fig. 18).

The S wave was a large wave that appeared just behind the Q wave in an
ECG, corresponded to the rise of aortic flow rate (F), its trough corresponded to
the vertex of the first derivative of aortic flow curve (dF/dt), and its amplitude
was correlated to the peak value of F, the peak value of dF/dt and the peak
value of the first derivative of aortic pressure (dPao/dt).

The acceleration given to the blood mass ejected from the left ventricle into
aorta is far greater than that from the right ventricle, and the ventricular im-
pulse is far stronger in the left ventricle.

Therefore, the role of the impulse generator is thought to be in the left
ventricle and the S wave is considered to be a vibration accompanying blood
ejection produced by the left ventricular contraction. The first derivative of
aortic flow (dF/dt), namely, acceleration of aortic flow reflects the force given to
the blood mass at the time of ejection. The differential of aortic pressure
(dPao/dt) is regarded to change in proportion to the aortic flow rate when
peripheral vascular impedance remains the same. Consequently, it may be
concluded that the S wave amplitude reflects the force given to the blood mass
ejected from the left ventricle, i.e. the force of ejection.

The V wave, corresponding to the third heart sound in the PCG, is con-
sidered to originate from the ventricular rapid filling.
A detailed report has been made on the transmission characteristics of the chest wall to the mechanical vibrations within the frequency range from 20 to 500 Hz. The transmission characteristics within the frequency range of the main waves of the A-VCGs, ranged 4 to 40 Hz, were examined in the present experiments. The fact that the transmission loss was approximately uniformly 6 dB in the range of 4 to 40 Hz and the A-VCGs from the chest wall seemed to reflect the vibration of ventricular wall with considerable fidelity.

Frequency distribution on the chest wall was approximately similar around the apex; this may suggest that only a normal degree of care be sufficient in selecting the site for transducer placement.

The A wave is considered to be a vibration accompanying atrial contraction. In the patients with ischemic heart disease, impaired ventricular distensibility caused by myocardial fibrosis and ventricular hypertrophy leads to increase in ventricular residual volume and rise in end-diastolic pressure. This results in increased resistance against end-diastolic rapid inflow by atrial contraction and consequently seems to be the reason for increased amplitude of the A wave. In fact, it has been pointed out by many workers that atrial gallop rhythm is often found in patients with coronary heart disease and with cardiac failure. Therefore, increase in the A wave amplitude and the A/S ratio with age in normal individuals, a significant increase in the A/S ratio in the angina pectoris group compared with normals in thirties, a significant increase in the A wave amplitude and the A/S ratio in the myocardial infarction group compared with normals may be explained by this mechanism.

In ischemic heart disease, lowering of cardiac contractility results in reduction in ventricular impulse and retarded acceleration, therefore decreased peak outflow. Consequently, the decreased amplitude of the S wave in the patients with myocardial infarction seems to reflect the lowered ejection rate and the reduced ejection force in an infarcted heart. The significant increase of the A/S ratio in angina pectoris cases seems also to be accounted for by the same way.

Exercise naturally increases heart rate and peripheral resistance. In normal individuals, increased amplitude of the S wave was observed directly after exercise, but there was no significant change in A wave amplitude. In angina pectoris cases, A wave amplitude increased and S wave amplitude remained unchanged or even decreased in some cases. This is thought to indicate that the lowered cardiac function which was not manifest at rest appeared clearly at the time of exercise and suggests that the A-VCG would offer a useful index for impaired ventricular reserve function.

Change accompanying inhalation of amyl nitrite in normal individuals was a slight increase in A wave amplitude, marked increase in S wave amplitude,
and decrease in the A/S ratio, but only a small change was observed in patients with angina pectoris. Amyl nitrite is said to increase cardiac output and venous return.\textsuperscript{52,53} The small changes seen in patients with coronary heart disease seem to indicate distinct expression of reduced myocardial reserve function.

Some of the disadvantages of the A-VCG are that the recording tends to become obscure when the subject is excessively obese and has a thick chest wall, and that the bed for recording must be considerably hard lest the precordial vibration should be damped.

This study indicates that the A-VCG is an easy and reliable clinical method capable of providing information quantitatively about the mechanical function of the heart.

**Acknowledgement**

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**References**