Kawasaki Med. J. Vol. 4: No. 2, 83-92, 1978

APEXCARDIOGRAPHIC INDEX "C-ΔACG" I. EXPERIMENTAL STUDIES

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Accepted for Publication on June 7, 1978

Abstract

Apexcardiographic index "C-ΔACG" was studied by animal experiments, and the results were demonstrated as follows.

1. The C point on ACG occurred almost simultaneously with a rise of the left ventricular pressure curve.

2. The peak of ΔACG preceded the peak dp/dt by 10.9 msec in average.

However, when contractility was altered in the same individual, it was suggested that the change of "C-ΔACG" correlated well with that of "t-peak dp/dt" and of contractility.

3. This parameter was prolonged after propranolol and was shortened after isoproterenolol.

4. While it was not possible to eradicate the effect of afterload, it was considered not possibly affected by preload.

This parameter, therefore, seems to be useful for evaluating the left ventricular contractility, particularly for detecting its changes within the same subject.

INTRODUCTION

Recently the first derivative of the apexcardiogram (ΔACG) has been used as one of the noninvasive methods for evaluating the left ventricular function[1-11].

Reale[1] and Vetter[2] have suggested that the interval from the onset of ventricular depolarization to the peak of ΔACG "t-ΔACG" is valuable in evaluating the left ventricular function. This interval "t-ΔACG", however, includes the electromechanical interval which differs from an individual to another, and it is prolonged markedly in left bundle branch block. For this reason, it seems that the left ventricular function can be represented more accurately by subtracting the electromechanical interval from "t-ΔACG".

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It is reported[12-14] that the C point on apexcardiogram (ACG) coincides well with the onset of the upstroke of the left ventricular pressure curve.

Therefore, the time interval from the C point on ACG to the peak of ΔACG " C-ΔACG " was studied in order to evaluate its efficacy and limitation by animal experiments.

MATERIALS AND METHODS

Fifteen mongrel dogs (10-25 Kg) were anesthesized with pentobarbital (30 mg/Kg), and the following procedures were introduced. High fidelity catheter-tipped micromanometers were inserted into the left ventricle from the right carotid artery, and into the aortic arch from the femoral artery.

The left ventricular pressure, its first derivative (dp/dt) and aortic pressure were simultaneously recorded with ACG and ΔACG in the left lateral decubitus position at a paper speed of 200 mm/sec. ACG was recorded using a transducer (TY303, Fukuda Electronic Co.) and ΔACG with RC circuit with time constant of 1 msec. Fig. 1 shows the measurement of " C-ΔACG ". After the above preparatory interventions isoproterenol (20-50 μg) and propranolol (10 mg) were administered intravenously to 10 dogs with intervals of 20 to 30 minutes in order to alter the contractility, and the correlation between " C-ΔACG " and the invasively derived parameters (V max, dp/dt/p (p=50 mmHg), max dp/dt and LVEDP) were studied. Of the four parameters obtained invasively, both V max (by Mason's method[15]) and dp/dt/p (p=50 mmHg) were measured by the developed pressure.

In order to observe the effects of preload and afterload, intravenous injection of 500 ml of dextran (10 dogs) and of 1 to 2 mg of methoxamine (7 dogs) were given.

In these instances the heart rate was kept constant by the right atrial pacing.

RESULTS

1. The time relation of the left ventricular pressure curve to ACG (Fig. 2)

1) Onset of the systolic upstroke.

The onset of the systolic upstroke of ACG (C-point) occurred almost simultaneously with the rise of left ventricular pressure curve, the apexcardiogram following the left ventricular pressure by a time difference of 2.4 msec$\pm$ 2.0 (mean$\pm$) 1 standard deviation).

2) First derivative

The peak of ΔACG always preceded the peak dp/dt by 10.9 msec$\pm$5.0 (mean$\pm$1 SD).

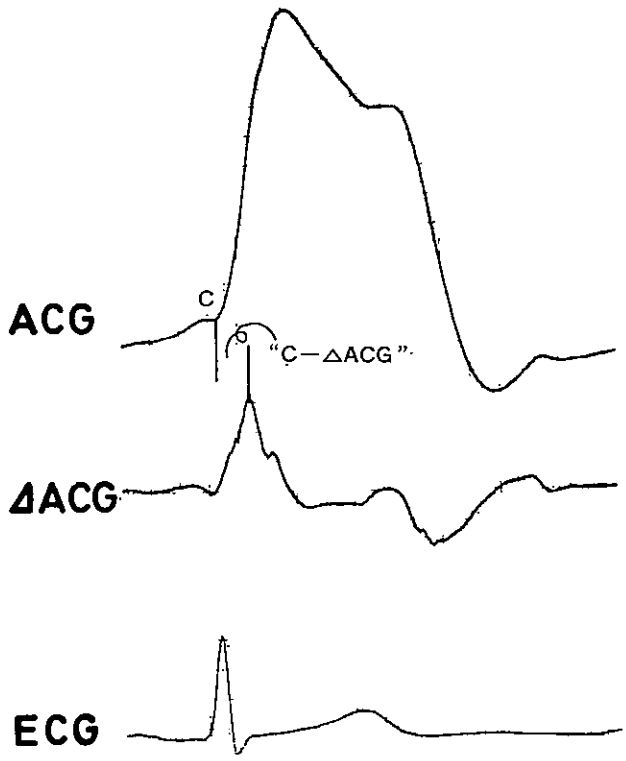

Fig. 1. The measurement of " C-⊿ACG " on apexcardiogram (ACG) and first derivative of ACG (⊿ACG). " C-⊿ACG " is the interval from the C point on AGG to the peak of ⊿ACG.

2. Alterations of the contractility

1) Relation of " C-ACG " to " t-peak dp/dt " (the interval from the onset of left ventricular pressure to peak dp/dt).

Fig. 3 shows a highly significant linear correlation between " C-⊿ACG " and " t-peak dp/dt " ($r=+0.87$) when contractility was changed.

2) Effect of changes in contractility on " C-⊿ACG "

Fig. 4 shows relationships of changes in " C-⊿ACG " to Vmax and to dp/dt/p ($p=50$ mmHg) at the time when contractility was changed. With isoproterenol Vmax and dp/dt/p ($p=50$ mmHg) were increased, while " C-⊿ACG " was shortened in every instances. In contrast, with propranolol Vmax and dp/dt/p ($p=50$ mmHg) were decreased and " C-⊿ACG " was increased in every instances.

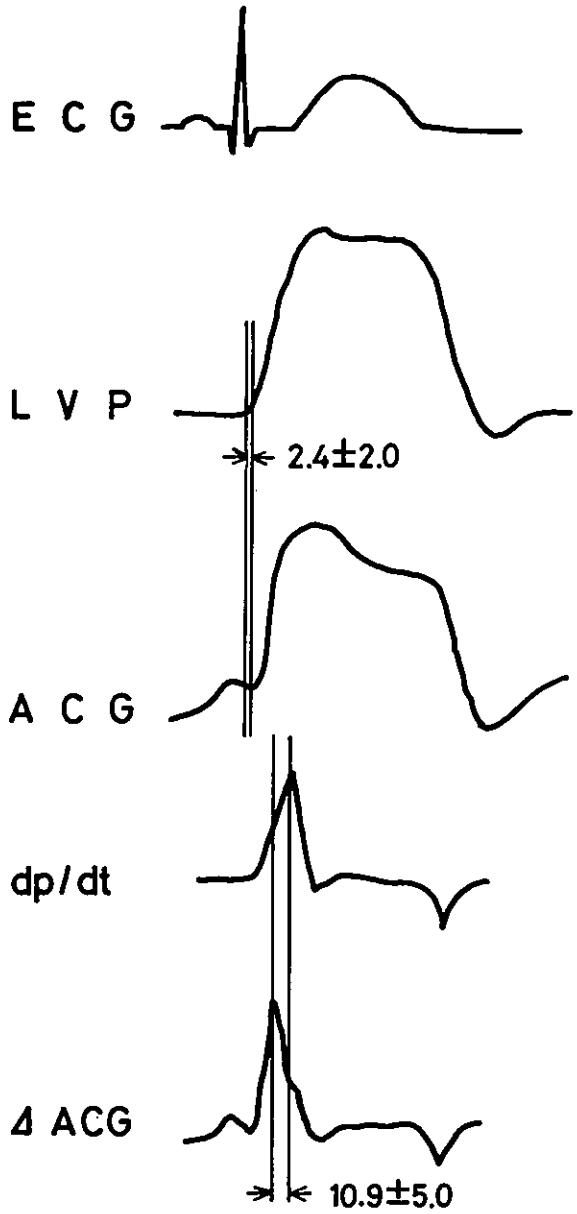

Fig. 2. The time relation of the left ventricular pressure curve to ACG. See the text.
Note LVP: left ventricular pressure
dp/dt: first derivative of LVP

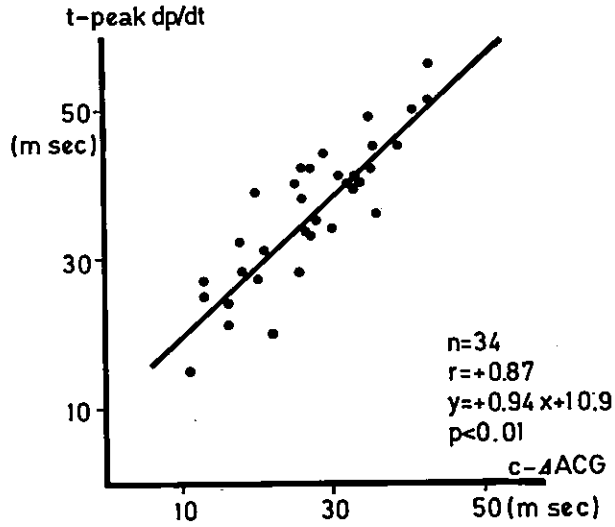

Fig. 3. Relation of "C-⊿ACG" to "t-peak dp/dt" when cotractility was altered.

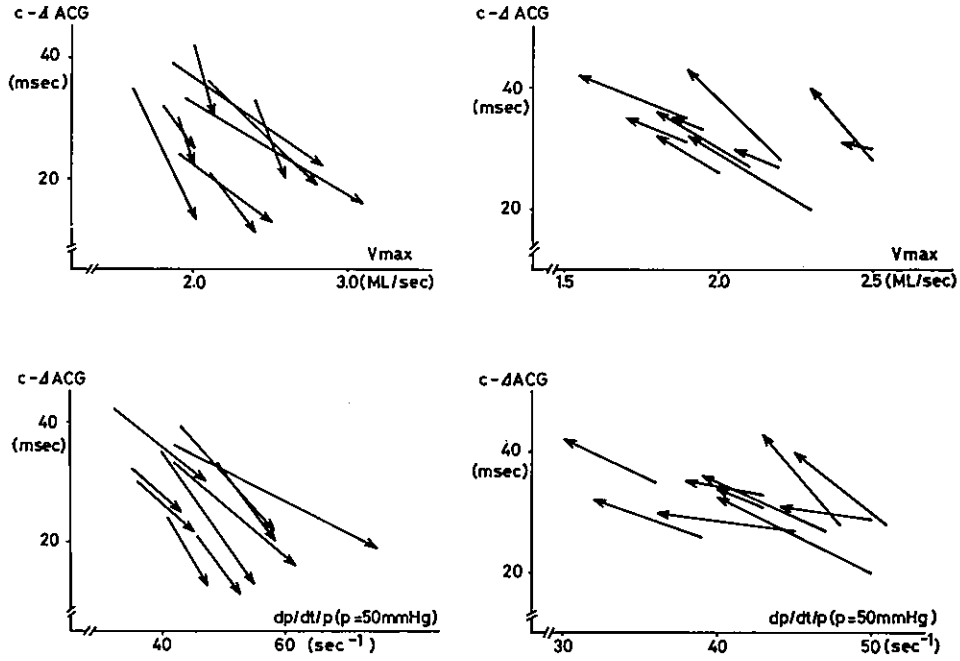

Fig. 4. Relation of changes in "C-⊿ACG" to Vmax and to dp/dt/p with propranolol and isoproterenol.
A : isoproterenol (20-50 ug)
B : propranolol (10 mg)

3) Relation of "C-⊿ACG" to various invasive parameters (Fig. 5) Close correlations were observed between "C-⊿ACG" and Vmax ($r=-0.83$), and dp/dt/p (p=50 mmHg) ($r=-0.81$). Less close but also significant correlations were observed between "C-⊿ACG" and max dp/dt ($r=-0.65$), and left ventricular enddiastolic pressure (LVEDP) ($r=+0.52$).

4) Comparison of "C-⊿ACG" with (peak DA/DT)/A (Table 1)

Both had approximately the same degree of correlation to Vmax, and to dp/dt/p (p=50 mmHg).

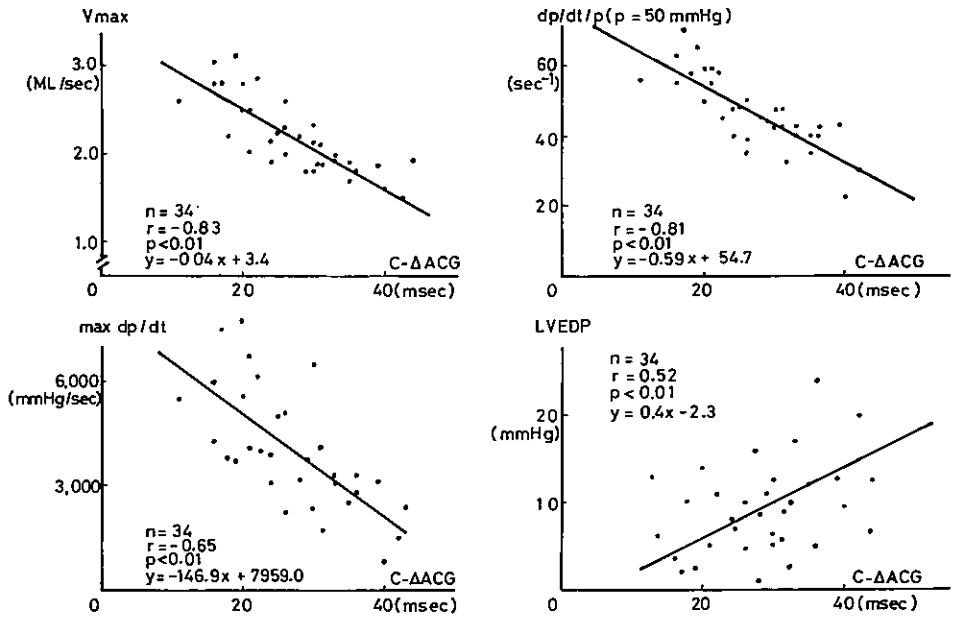

Fig. 5. Relation of "C-⊿ACG" to various invasive parameters with alteration of contractility.

TABLE 1. Correlation coefficient (r value) between noninvasive parameters [(peakDA/DT)/A, C-⊿ACG] and invasive ones [Vmax, dp/dt/p (p=50mmHg)]

invasive \ non invasive	(peak DA/DT)/A	C-⊿ACG
Vmax	r=0.83	r=−0.83
dp/dt/p (p=50mmHg)	r=0.85	r=−0.81

3. Alterations of the afterload (Fig. 6-A)

After an intravenous injection of 1 to 2 mgs of methoxamine the aortic diastolic pressure rose by 21 mmHg in average and " C-⊿ACG " was shortened by 6 % in average, being not significant. When the aortic diastolic pressure rose by 35 mmHg in average, " C-⊿ACG " was shortened significantly by 9.5 % in average ($p < 0.05$).

4. Alterations of the preload (Fig. 6-B)

While with a rapid infusion of 500 ml of dextran the LVEDP rose by 17 mmHg in average, " C-⊿ACG " did not show any significant difference from the control. With dextran infusion the aortic diastolic pressure showed a slight fall in general (5 mmHg in average).

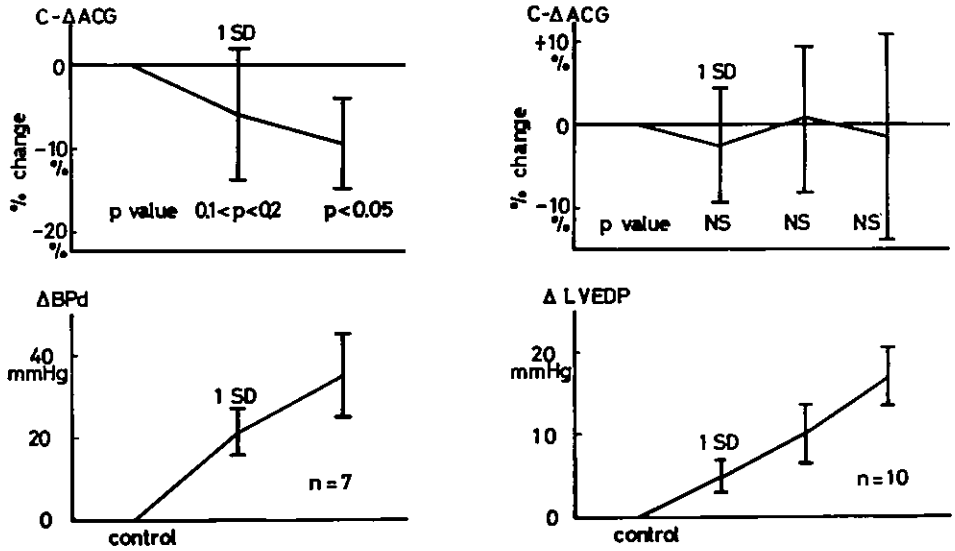

Fig. 6. Percent change from control to post-intervention values in " C-⊿ACG ".
A : Alteration of afterload by 1-2 mg of methoxamine.
B : Alteration of preload by 500 ml of dextran.
Note Bpd: diastolic blood pressure
NS: non-significant
1 SD: 1 standard deviation

DISCUSSION

The time interval " t-peak dp/dt " represents the duration of the development of maximal tension during isovolumic contraction phase of the left ventricle. The concept of " t-peak dp/dt " was proposed by Reeves *et al.*[16] which was later developed by Mason *et al.*[17] This parameter is reported to

serve as one of the parameters representing the left ventricular contractility[18-21]. Reale[1] and Vetter[2] proposed the time interval "t-ΔACG" to measure "t-peak dp/dt" noninvasively.

The "C-ΔACG" interval, used at present, is the interval by subtracting electro-mechanical interval from "t-ΔACG", and it seems to be more theoretical to represent the left ventricular contractility as discussed earlier.

The C point on ACG is reported to coincide approximately with the onset of upstroke of the left ventricular pressure curve[12-14]. The results are also in accordance with theirs, suggesting that the C point on ACG may serve as an indicator of onset of the left ventricular systolic upstroke.

Whether or not the peak of ΔACG and the peak dp/dt coincide are controversial. Reale *et al.*[1] state that the peak of ΔACG approximately coincides with peak dp/dt. On the other hand, Willems[12] with dog study and Manolas[13,14] with human experiment reported that the peak of ΔACG preceded the peak of dp/dt by 10 to 20 msec. In the present experiment the peak of ΔACG always preceded the peak of dp/dt by the average of 10.9 msec. However, in the correlation between "t-peak dp/dt" and "C-ΔACG" at the time when the contractility was altered, the two showed a positive correlation as high as $r=+0.87$. "C-ΔACG" was shortened after the administration of isoproterenol and prolonged after propranolol. In addition, "C-ΔACG" revealed a highly negative correlation to Vmax ($r=-0.81$) and to dp/dt/p ($r=-0.79$), which is considered to represent the cardiac contractility.

Although the time interval "C-ΔACG" did not coincide with "t-peak dp/dt", it has been demonstrated that in the same subject when contractility is altered the change of "C-ΔACG" coincides well with that of "t-peak dp/dt" and of contractility. This parameter, therefore, seems to be particularly useful for detecting changes in the left ventricular contractility within the same individual.

The correlation coefficient of "C-ΔACG" to Vmax and to dp/dt/p corresponded approximately to that of "(peak DA/DT)/A" as reported previously[9]. While the calibration is necessary in calculation of "(peak DA/DA)/A" it is not in "C-ΔACG". Therefore, the latter seemed to be more simple index in evaluating the left ventricular contractility.

Finally, a relation of "C-ΔACG" to preload and afterload was evaluated. This parameter seemed to be affected by afterload as shown in Fig. 6-A. When LVEDP rose by 17 mmHg in average after dextran, this parameter did not change significantly, and the fall of aortic diastolic pressure was slight, suggesting that this parameter may not be affected by preload.

Acknowledgment

I am greatly indebted to Dr. Toshitami Sawayama, Professor of Medicine, Kawasaki Medical School, for his participation in this study.

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