Ventriculoarterial Coupling in Normal and Failing Heart in Humans

Hidetsugu Asanoi, Shigetake Sasayama, and Tomoki Kameyama

To investigate coupling between the heart and arterial system in normal subjects and cardiac patients, we determined both the slope of the left ventricular end-systolic pressure-volume relation (ventricular elastance) and the slope of the arterial end-systolic pressure-stroke volume relation (effective arterial elastance) in three groups of subjects: group A, 12 subjects with ejection fraction of 60% or more; group B, seven patients with ejection fraction of 40-59%; and group C, nine patients with ejection fraction of less than 40%. We also determined the left ventricular stroke work, end-systolic potential energy, and the ventricular work efficiency defined as stroke work per pressure-volume area (stroke work + potential energy). In group A, ventricular elastance was nearly twice as large as arterial elastance. This is a condition for a maximal mechanical efficiency. In group B, ventricular elastance was almost equal to arterial elastance. This is a condition for maximal stroke work from a given end-diastolic volume. In group C, ventricular elastance was less than one half of arterial elastance, which resulted in increased potential energy and decreased work efficiency. Thus, the present study suggests that ventriculoarterial coupling is normally set toward higher left ventricular work efficiency, whereas in patients with moderate cardiac dysfunction, ventricular and arterial properties are so matched as to maximize stroke work at the expense of the work efficiency. Neither the stroke work nor the work efficiency is near maximum for patients with severe cardiac dysfunction.

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The ventricle is a generator of hydraulic energy, which transfers the mechanical energy of contraction to the blood accumulated in the ventricular chamber. The stroke work (SW) and power output from a given end-diastolic volume are strongly influenced by the input impedance of the arterial system. In physics and engineering, an energy source and its load are considered matched when a maximal amount of energy is transferred from the source to the load. The matching occurs when the input impedance of the load equals the output impedance of the energy source. Using isolated heart preparations, several investigators confirmed the validity of this matching concept for the coupling of the real ventricle with simulated arterial loads. That is, they verified that the SW was maximized when the afterloaded impedance was close to the internal impedance of the ventricle. This does not mean, however, that the physiological control mechanisms indeed adopt this criterion for optimal coupling between the heart and artery under physiological circumstances in vivo.

Another criterion for optimal coupling between an energy source and its load is the principle of economical fuel consumption, or mechanical efficiency. In the case of heart, the mechanical efficiency is defined as the ratio of SW to myocardial oxygen consumption per beat (MVO₂). Several investigators looked into this efficiency of cardiac pump. They came to a similar theoretical conclusion that the mechanical efficiency from a given end-diastolic volume becomes maximal when arterial impedance is nearly one half the cardiac output impedance. Again, it is not known at all whether the physiological controller of the heart and arterial system prefers this efficiency-oriented criterion at rest and the work-maximization criterion in stressful conditions.

In the present study, therefore, we investigated the resting human's matching of the ventricular properties quantified by the slope of end-systolic pressure-volume (P-V) relation (E₉₀) with arterial load properties expressed by the slope of end-
systolic pressure-stroke volume (P-SV) relation (\(E_s\)) under normal and variably depressed cardiac conditions, with special reference to those two matching principles described above. That is, we estimated \(E_{sv}, E_s, SW, MVO_2,\) and total mechanical energy released per contraction from the P-V data and studied how the relative magnitudes of \(E_{sv}\) and \(E_s\) are in these subjects with different degrees of cardiovascular stress.

**Subjects and Methods**

Eight normal subjects (aged 19–62 years; mean, 32.0 years) with no symptoms and signs of cardiac

**TABLE 1. Individual Data of Left Ventricular and Arterial End-Systolic Pressure-Volume Relation**

<table>
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Based on resting left ventricular ejection fraction, subjects were divided into three groups: group A, ejection fraction ≥60%; group B, ejection fraction 40–59%; and group C, ejection fraction ≤39%. EDVI, end-diastolic volume index; ESVI, end-systolic volume index; SVI, stroke volume index; EF, ejection fraction; ESP, end-systolic pressure; PCW, pulmonary capillary wedge pressure; HR, heart rate; \(V_0\) and \(E_{sv}\), volume intercept and slope of left ventricular end-systolic pressure-volume relation; \(E_s\), slope of arterial end-systolic pressure-volume relation; SD, standard deviation of the mean; NS, not statistically significant.

Subjects

Eight normal subjects (aged 19–62 years; mean, 32.0 years) with no symptoms and signs of cardiac
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FIGURE 1. Simultaneous recordings of left ventricular echocardiogram and direct arterial pressure. Left ventricular volume was determined by the formula of Teichholz et al., and ventricular end-systolic pressure was approximated from the arterial dicrotic pressure. EDD, end-diastolic dimension; ESD, end-systolic dimension; ESP, end-systolic pressure.

Subjects were divided into three groups based on their resting left ventricular ejection fraction (EF) determined by echocardiography. Group A consisted of 12 subjects with left ventricular EF of 60% or more. Group B consisted of seven patients with mild left ventricular dysfunction in whom EF was 40–59%. Group C consisted of nine patients with more marked left ventricular dysfunction in whom EF was less than 40%.

Symotic Pressure and Left Ventricular Volume

All patients were in regular sinus rhythm and were studied in the supine postabsorptive state. A 19-gauge cannula was inserted percutaneously into a brachial artery and was connected to a strain-gauge manometer (model P50, Spectramed, San Juan, Puerto Rico). After control recordings at rest, phenylephrine (5 mg/100 ml) was infused intravenously to increase systolic pressure in gradual increments of approximately 20 mm Hg. Subsequently, adequate recovery time was allowed for peak systolic pressure to return to the baseline level, and then a sodium nitroprusside infusion (50 mg/500 ml) was started to decrease systolic pressure by about 20 mm Hg. Thus, the systolic pressure was changed by about 40 mm Hg during these interventions. Two-dimensional targeted M-mode echocardiograms of left ventricular cavity were recorded by a
Toshiba SHA-60A with a 3-MHz transducer (Toshiba, Japan) used simultaneously with arterial pressure (Figure 1). All data were recorded at a paper speed of 50 mm/sec. The end-diastolic diameter was obtained at the peak of the R wave of the electrocardiogram, and the end-systolic diameter at the initial component of the second heart sound. Left ventricular volume was determined with the formula of Teichholz et al.\textsuperscript{12}

Left ventricular end-systolic pressure was approximated from the arterial dicrotic pressure, which is considered to be caused by aortic valve closure. It was clearly discernible on pressure wave form in all patients.

**Matching Analysis**

Ventriculoarterial matching was analyzed in the framework recently developed by Sunagawa et al.\textsuperscript{13,14} and Burkhoff and Sagawa.\textsuperscript{11} Namely, the ventricular output impedance properties were quantified in terms of $E_a$. The arterial input impedance properties were expressed in terms of the effective arterial elastance, $E_a$.

**Ventricular end-systolic pressure-volume relation.** It has been shown that the ventricular end-systolic P-V relation is linear, and the ventricular contractile properties can be quantified primarily by its slope, $E_{es}$, with the aid of its volume axis intercept, $V_o$.\textsuperscript{15-17} To obtain the end-systolic P-V relation, we plotted more than five different dicrotic arterial pressures against corresponding left ventricular end-systolic volumes during the pharmacological pressure manipulation in each subject. Then, linear regression of these pressures on the volumes was performed to determine $E_{es}$ (Figure 2A).

**Arterial end-systolic pressure-stroke volume relation.** Given a constant heart rate, arterial end-systolic pressure changes with stroke volume in a roughly linear relation (Figure 2B). The slope of this relation is in proportion to the impedance that the arterial tree offers to the stroke flow. Thus, the arterial properties can be represented as a first approximation by the slope of the arterial end-systolic P-SV relation. Sunagawa et al\textsuperscript{13} called this slope effective arterial elastance, $E_a$. We superimposed this arterial end-systolic P-V relation on the ventricular end-systolic P-V relation in the same P-V plane by transposing the stroke volume axis of the arterial end-systolic P-SV relation line and letting its origin fall on the end-diastolic volume point of the ventricular end-systolic P-V relation line (Figure 2C). This superposition enables a graphical coupling of the ventricular output properties with the arterial input impedance properties. The equilibrium end-systolic pressure and volume that should exist when the ventricle is coupled with the arterial system can be obtained as the intersection between these two end-systolic P-V relation lines (Figure 2C). Conversely, it can be seen in Figure 2C that if we connect the equilibrium point (I) with end-diastolic volume, the slope of this line represents $E_a$.

**FIGURE 2.** The framework of analysis for coupling the ventricle with the arterial load. The mechanical characteristics of the left ventricle are expressed by the end-systolic pressure (Pv)-volume (V) relation (A). The mechanical characteristics of the arterial system are expressed by the arterial end-systolic pressure (Pa)-stroke volume (SV) relation (B). The arterial Pa-V relation can be superimposed on the ventricular Pv-V relation in the same pressure (P)-volume (V) plane. The equilibrium end-systolic pressure and volume when the ventricle is coupled with the arterial system are obtained from the intersection (I) between these two P-V relation lines (C). Closed circles, baseline state; open circles, phenylephrine; open triangles, nitroprusside. $E_{es}$ and $V_o$, slope and volume axis intercept of ventricular end-systolic pressure-volume relation; EDV, end-diastolic volume; $E_a$, slope of arterial end-systolic pressure-stroke volume relation.
Stroke Work and Work Efficiency

Ventricular P-V loop area accurately represents SW of the ventricle. For simplicity, we assumed that the P-V loop could be regarded as a rectangle whose height was end-systolic pressure and whose width was stroke volume. Under the simplifying assumption, SW was calculated as end-systolic pressure x (end-diastolic volume - end-systolic volume). End-systolic pressure and end-systolic pressure-volume relation are the graphically determined data points at which two end-systolic P-V relation lines intersect with each other (Figure 3). The values of SW, Ees, and Ea in three groups were surveyed in reference to the theoretical and experimental analyses by Sunagawa et al.\(^8\) that SW was maximized with Ees = Ea. As an index of the efficiency of ventricular contraction, we studied the ratio of SW to the systolic P-V area (PVA), which has been defined by Suga\(^8\) as the total mechanical energy of ejecting stroke volume relation (Ea). Closed circle, baseline state; open circles, phenylephrine; open triangles, nitroprusside; ESPf calculated end-systolic pressure; Vo, volume axis intercept of ventricular end-systolic pressure-volume relation; ESVf calculated end-systolic volume; EDV, end-diastolic volume.

Myocardial Oxygen Consumption

Recently, Suga and his colleagues\(^18,19\) have found that PVA linearly correlates with MVO\(_2\) per beat. This relation is characterized by a slope (a) and an intercept (b) as MVO\(_2\) = a x PVA + b. However, when basal inotropic state differs among individual cases, PVA cannot be an alternative of MVO\(_2\) because of the difference in intercept b.\(^20,21\) Accordingly, we tried to calculate actual mechanical efficiency by measuring myocardial oxygen consumption from thermodilution coronary sinus flow and arterial-coronary sinus oxygen difference. This measurement was available among 12 of 28 patients (four in group A, three in group B, and five in group C). In eight subjects, these two measurements were obtained separately, but all measurements of myocardial oxygen consumption were performed within 2 days after determination of SW at the same resting heart rate and blood pressure.

Proximal coronary sinus catheterization was performed with a dual thermistor thermodilution catheter (Webster Laboratories Incorporated, Alameda, California). Thermodilution coronary sinus blood flow was determined in duplicate with standard technique\(^22\); the proximal thermistor was carefully placed in the ostium of the coronary sinus. Position was confirmed by both fluoroscopic appearance throughout the study and hand injection of small amounts of contrast medium. Arterial and coronary sinus blood samples were drawn simultaneously for the determination of oxygen saturation. Blood oxygen content was calculated as the product of the percent oxygen saturation and the oxygen-hemoglobin binding capacity. Myocardial oxygen consumption was calculated as the product of the coronary sinus flow and the arterial-coronary sinus oxygen difference.

Since SW and myocardial oxygen consumption are the energy units expressed by mm Hg x ml and ml O\(_2\), respectively, these units were converted into a common unit of energy, the joule (J), with the following conversions: 1 mm Hg x ml = 1.33 x 10\(^{-4}\) J, 1 ml O\(_2\) = 20J. Mechanical efficiency was expressed conventionally by (SW x heart rate)/myocardial oxygen consumption. Since Burkhoff and Sagawa\(^11\) predicted that the maximal mechanical efficiency of ventricular contraction from a constant preload is gained when Ees = Ea/2, we looked at the ratio of Ea to Ea with this theoretical conclusion in mind.

Methodological Considerations

Reproducibility for echocardiographic volume. The intraobserver differences for 23 duplicate measurements of left ventricular volume were 0.3±4.2 ml (r = 0.99, p < 0.001) for end-diastolic volume and 0.2±2.6 ml (r = 0.99, p < 0.001) for end-systolic volume, respectively. The interobserver differences for duplicate studies were 4.1±4.8 ml (r = 0.99,
RESULTS

Baseline Cardiac Function

The data are listed for all subjects in Table 1. There was no significant difference in heart rate and end-systolic pressure among the three groups. End-diastolic volume increased with reduction in EF, but only differences between group A and group C reached the statistical significance. End-systolic volume was also greater in group C than in group A and group B. Stroke volume in group C was significantly less than in group A and group B. Pulmonary capillary wedge pressure was elevated in only six patients in group C, but this elevation was not greater than 20 mm Hg, except in one patient.

Ventricular and Arterial Properties

End-systolic pressure was elevated by 20±8 mm Hg with phenylephrine and decreased by 18±8 mm Hg with nitroprusside. Alterations in heart rate during pressure changes were 13±8 beats/min. A linear relation between corresponding end-systolic pressure and end-systolic volume was observed in all patients during afterload change (Table 1). Representative P-V data and end-systolic P-V relations for patients of each group are shown in Figure 5. Volume axis intercept (V_o) in group C was significantly greater than in group A and group B. E_{es} significantly differed between group A and group C but not between group A and group B and between group B and group C. The effective arterial elastance, E_a, showed an increase with reduction in EF, and the difference between group A and group C was statistically significant.

The ratio of E_a to E_{es} (E_a/E_{es}) showed a progressive increase as EF decreased. This ratio in subjects in group A was significantly lower than those with marked ventricular dysfunction (group C), but the differences between group A and group B and between group B and group C were not statistically significant.

Left Ventricular Work

Individual data related to SW and PVA are listed in Table 2. SW tended to be reduced in group C as compared with that in group A and group B, but the differences did not reach statistical significance. End-systolic potential energy in patients with cardiac dysfunction (group B and group C) was significantly greater than in group A. There were no significant differences in the PVA among three groups. The ratio of SW to total mechanical energy (PVA) was highest in group A and progressively decreased with the reduction of EF; significant differences existed among the three groups.

We also calculated the ratio of SW to SW_{max} for a given end-diastolic volume, where SW_{max} is the maximal SW obtainable if E_a were equal to E_{es}. 

Statistical Analysis

Data are expressed as mean±SD. The statistical significance of differences in hemodynamic variables among three groups was tested by analysis of variance, and multiple comparisons were made by the Bonferroni method. Values of p<0.05% were considered to represent a statistical significance.

FIGURE 4. Relation between echocardiographic (ECHO) and contrast ventriculographic (ANGIO) volume in 75 tracings. There is a good correlation between the two measurements.

$p<0.001$ at end diastole and 3.9±3.3 ml ($r=0.99$, $p<0.001$) at end systole.

The correlation between 73 left ventricular volumes encompassing a wide range of volumes (44 end-diastolic volumes, 29 end-systolic volumes) determined by echocardiography and contrast ventriculography was 0.92 ($p<0.01$). However, the echocardiographic method slightly underestimated left ventricular volume (Figure 4), presumably due to underestimation of the long axis of left ventricle.

Left ventricular end-systolic pressure. To validate the use of dicrotic pressure of the brachial artery as a measure of left ventricular end-systolic pressure, we compared left ventricular pressure obtained by micromanometer-tip catheter and brachial arterial pressure in groups of patients with different contractile state (six patients with EF<40% [group 1] and 12 patients with EF≥40% [group 2]). The comparison was also made at several pressure levels during phenylephrine infusion in the single subject. The dicrotic arterial pressure was lower than the left ventricular end-systolic pressure by 2.6±1.6 mm Hg in group 1 and by 3.7±2.1 mm Hg in group 2. However, these differences were so slight as to be almost negligible relative to the pressure changes for the determination of the ventricular end-systolic P-V relation. When vascular tone was changed by phenylephrine in four patients, dicrotic arterial pressure (y) changed linearly with left ventricular end-systolic pressure (x) so that $y=1.0x−0.6$, $r=0.98$ ($p<0.001$).
Mechanical efficiency tended to be decreased in group C; the averages were 30±3% in group A, 24±6% in group B, and 21±6% in group C, but these changes did not reach statistical significance.

Discussion

To the best of our knowledge, this is the first study in humans where the coupling between the ventricular pump and arterial afterload is investigated in terms of end-systolic elastance of the ventricle and effective input elastance of the arterial tree. In group A, with normal EF, the ventricle was in a good contractile state with Ees of 4.5±2.0 mm Hg/ml/m³ and EF of 60% or more. We found that their Ees was always set lower than Eas and resulted in a greater work efficiency and a smaller SW for the given contractility Ees and preload than when Ees was equal to Eas. In those patients whose ventricles were in a mildly depressed state with Ees of 2.5±1.1 mm Hg/ml/m³ and EF of 50%, Ees was found to be nearly equal to Eas. With this Ees/Eas ratio, their ventricles were performing at almost maximal SW possible at a given preload. In those patients whose ventricles were severely depressed with Ees of 1.5±0.7 mm Hg/ml/m³ and EF of less than 40%, the Ees/Eas ratio (2.56±2.03) was substantially higher than in the normal group (0.46±0.17) and the mildly depressed heart group (0.90±0.21).

Left Ventricular Mechanical Efficiency

Table 3 listed the individual data of myocardial oxygen consumption and mechanical efficiency.
ventricular compliance was reduced with elevated end-diastolic pressure. All patients in the present study were free from any sign of pulmonary congestion at rest. The average of mean capillary wedge pressure was 15 mm Hg even in group C with marked left ventricular dysfunction. Therefore, the errors in the SW estimated could not be extremely large.

Nine of 28 subjects showed negative values of $V_{o}$. The physiological meaning of the $V_{o}$ of the end-systolic P-V relation is uncertain. There are several possible explanations. First, the end-systolic P-V data. A third and related possibility is that $V_{o}$ relates the V-P relation is not linear in the regions of the $V_{o}$-$V_{Q}$. Nine of 28 patients showed negative values of $V_{o}$. The physiological meaning of the $V_{o}$ of the end-systolic P-V relation is uncertain. There are several possible explanations. First, the end-systolic P-V relation is not linear in the regions of the $V_{o}$-$V_{Q}$.
might be affected by changes in afterload used in the present study.26

A number of animal studies6–8,13,27,28 have recently been performed to answer the question: what is the optimal coupling between the ventricle and arterial load actually operative in vivo under physiological and pathological circumstances? Almost all of these experimental studies produced a similar answer that physiological states of the ventricle and arterial input impedance are matched to produce a maximal SW or power. Wilcken et al27 studied the effects of sudden changes in arterial input impedance in anesthetized and conscious dogs and found that either increase or decrease in the impedance reduced the stroke power and work. Sunagawa et al6 have theoretically predicted and experimentally validated in isolated physiologically loaded canine heart that maximization of left ventricular SW occurs when ventricular contractility measured by E\textsubscript{max} and the simulated arterial input impedance expressed by E\textsubscript{a} are matched to equal each other (Figure 3). Recently, van den Horn et al7 demonstrated in the open-chested anesthetized cat that, at given arterial pressure and cardiac output (therefore, ventricular power) required by the body, the ventricular properties (contractility) are adjusted to produce the required power at the peak of its power-flow relation curve. More recently, Myhre et al29 also showed that in anesthetized and open-chested dog’s heart, the normal ventricle yielded maximum SW against normal arterial afterload, but during acute depression of left ventricular performance, the working point of the ventricle shifted from the top of the dome-shaped SW-stroke volume relation curve onto the left limb. These conclusions on “physiological” matching disagree with the present findings in normal subjects. Rather, we found the ventriculoarterial coupling to be set for a maximal SW in group B patients with mildly depressed heart function. This discrepancy may be explained by remembering that, in these studies (except part of Wilcken’s study27), the animals were anesthetized and open-chested and that, in many of them, the heart was excised. Under the circumstances, the ventricular contractility could be significantly less than physiological. At the same time, the physiological values of arterial input impedance taken from the literature might be from animals anesthetized and with reflexly augmented vascular tone.

There are other studies,1,10,11 experimental and theoretical, which reached conclusions quite consistent with the present findings. First, Elzinga and Westerhof10 showed in isolated cat heart preparation that the maxima of mean external power, oxygen consumption, and mechanical efficiency of ventricular contraction were achieved at different operating points on their ventricular pump function curve. From the P-V data obtained in the isolated cat ventricle, Piene and Sund1 calculated the pump efficiency as we did in the present study and found that right ventricular pump efficiency became maximum when pulmonary impedance values loaded on

### Table 3. Individual Data of Myocardial Oxygen Consumption and Mechanical Efficiency

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<th>CSF (ml/min)</th>
<th>A–CSDO\textsubscript{2} (ml/100 ml)</th>
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CSF, coronary sinus flow; A–CSDO\textsubscript{2}, arterial-coronary sinus oxygen difference; MVO\textsubscript{2}, myocardial oxygen consumption; SW, left ventricular stroke work; HR, heart rate; SD, standard deviation of the mean.
the ventricle were in the physiological range. Finally, Burkhoff and Sagawa\textsuperscript{11} theoretically analyzed the arterial load that would produce maximal SW and maximal mechanical efficiency of ventricular contraction. That is, they showed that the coupling condition for maximal SW is $E_a = E_{ca}$ whereas that for maximal efficiency is $E_a = E_{ca}/2$. They further speculated that the earlier concept on the physiological coupling between the ventricular pump and arterial afterload might have been misguided by the nonphysiologically low $E_a$ value and high total peripheral resistance value determined in the surgically compromised state.

These findings were surprisingly similar to the present findings. In the present study, the normal coupling condition in group A was nearly $E_a = E_{ca}/2$, as predicted by Burkhoff and Sagawa\textsuperscript{11} to achieve maximal mechanical efficiency. In moderately depressed heart, the ventricle and arterial system were matched to maximize SW ($E_a = E_{ca}$). These hearts generate a greater potential energy with resultant reduction in work efficiency of the left ventricle as compared with normal hearts. Work efficiency (SW/PVA) is a monotonically decreasing function of $E_a/E_{ca}$, $SW/PVA = 1/[1+ (E_a/E_{ca})^2]$. On the other hand, the relation of $MV_{O2}$ to PVA is crucially influenced by the inotropic state ($E_a$). Suga and coworkers\textsuperscript{18,19} have demonstrated the linear relation between $MV_{O2}$ and PVA. This relation has a nonzero positive intercept for PVA=0 ($MV_{O2} = a \times PVA + b$, $b > 0$). They also showed that when the inotropic state is enhanced, the $MV_{O2}$/PVA relation shifts upward (increase in b) and when the inotropic state is depressed, the relation shifts downward (decrease in b).\textsuperscript{20,21} Consequently, with the depression of contractile state, PVA/$MV_{O2}$ becomes increased for a given PVA. In moderately depressed heart, mechanical efficiency did not decrease to the extent as SW/PVA did, presumably due to an increase in PVA/$MV_{O2}$. In contrast, severely depressed hearts, as shown in group C patients, could no longer maintain SW properly and result in the mismatch in terms of mechanical efficiency probably due to the substantial fall in work efficiency.

The nature of the error detector for ventriculoarterial coupling is unclear. It cannot be explained by the receptors that give out an output proportional to mechanical efficiency or stroke power as in the regulation of mean arterial pressure and flow. Therefore, the efficiency or power matching might be regarded as a coincidence that resulted from cardiovascular adjustment to achieve physiological arterial pressure and flow.

We tried to extend experimental observations on optimal ventriculoarterial coupling to humans. The present study delineated the distinctive aspect between normal and variably depressed left ventricle in terms of the matching concept. The results of our study suggest that this conceptual framework for quantifying ventriculoarterial interaction has great potential usefulness to help us gain insight into the relevance of adaptational changes in congestive heart failure.

Acknowledgments

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References


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