Cardiac Hemodynamics During Stimulation of the Right Atrium, Right Ventricle, and Left Ventricle in Normal and Abnormal Hearts

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In previous reports we and others have described the hemodynamic consequences of varying the rate of artificial pacing of the right ventricle in patients with complete heart block. Since the wires of a permanent pacemaker for treatment of complete heart block are implanted in the left ventricle and previous data were obtained from right ventricular pacing, it becomes important to determine whether the site of implantation of pacemakers is a critical determinant of cardiac output in man.

Experimental studies in dogs by Fletcher and associates with electrodes implanted in the apex, the high lateral wall, and the posterior septal region of the left ventricle have shown no significant differences in cardiac output and mean arterial pressure. Lister and co-workers, however, have demonstrated in dogs that the pacemaking site significantly influences cardiac output. Since this information has been available only in dogs, we have undertaken to study in man (1) the influence of pacemaking sites on the hemodynamics of normal subjects, in patients with compensated heart disease, and in patients with heart failure, and (2) the contribution of atrial systole to the cardiovascular dynamics of the normal and the abnormal heart.

Methods

A group of 26 persons was studied. Twenty-four had normal sinus rhythm, one had congenital complete heart block, and one had atrial fibrillation. Included in the group were 11 normal subjects, seven patients with documented coronary artery disease (postmyocardial infarction with or without angina pectoris), two with idiopathic myocardial hypertrophy, and one each with essential hypertension, mild mitral stenosis, mild mitral insufficiency, congenital complete heart block, idiopathic bradycardia, and postoperative constrictive pericarditis. Ages ranged from 15 to 74 years with a mean of 47 years. There were 14 males and 12 females.

The studies were performed under local anesthesia with mepivacaine hydrochloride (Carbocaine 1%) and with meperidine (Demerol), 50 mg., as premedication. A no. 8 Zucker bipolar electrode catheter was introduced into the right cubital vein and advanced to the right ventricle. The right brachial artery was exposed and a second no. 8 Zucker bipolar electrode catheter was introduced into the artery and advanced to the left ventricle. A no. 18 Cournand needle was introduced into the right femoral artery for measurement of arterial pressure and for sampling of the indicator. The wires of the electrode catheter were connected with an external transistorized pacemaker (Electrodyne, model TR 3) for control of the heart rate. This unit has been modified to provide faster rates of pacing. The range of rate could thus be varied from 60 to 180 per minute. This pacemaker delivers a nonphasic round-topped DC pulse of between 2 and 3 msec in duration and its voltage can be varied from 0 to 15 volts. Right ventricular pressure or right atrial pressure, left ventricular pressure, and femoral artery pressure were measured simultaneously with a PA3Db Statham pressure transducer. The first derivative of the femoral artery pressure was measured using an R/C differentiating circuit. Mean arterial pressure was obtained by electronic filtering. Heart rate was measured from lead II of the electrocardiogram simultaneously recorded with the dye curve. Ejection time was measured from the indirect carotid artery tracing by using a Sanborn pulse

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wave crystal microphone (model no. 374). Peripheral resistance, ventricular power, mean systolic ejection rate, stroke power, and stroke work were calculated from standard formulas. Peak systolic pressure was used in the formula of Sarnoff and associates\textsuperscript{13} for calculation of the tension-time index. All pressures, dp/dt, carotid tracings, and electrocardiograms were simultaneously recorded in the eight-channel Dr-8 oscillographic photographic recorder* at a paper speed of 200 mm/sec with 20-msec time lines. A minimum of six consecutive beats was recorded, and measurements were made for each heart beat and averaged.

The cardiac output was determined with the indicator-dilution technique using indocyanine green as an indicator. A precalibrated dye-dilution tube (BD-X 412 LST) with a one-way stopcock was filled with 6.25 mg of dye. The indicator was rapidly injected into the right ventricle or right atrium, and the tube was flushed with 10 ml of 5% dextrose in water. A 50 ml lubricated heparinized syringe was placed on a withdrawal infusion pump (Harvard Instruments, model 600-900), and blood was withdrawn from the femoral artery at a constant rate of 38.2 ml/min. A Gilford cuvette densitometer (model 130 IR) was used to detect the injected dye. After the dye curves had been inscribed, the blood was rein fused into the patient's arterial system. Cardiac output was obtained in duplicate and averaged. The output of the densitometer was connected to a dye-dilution computer (Sanborn model 130), and cardiac output was calculated by this means.\textsuperscript{14} At the end of the procedure the response of the densitometer to two concentration standards was obtained by using the same flow rate and densitometer attenuation. Zero and two calibration values of 2 and 4 mg of dye per liter of blood were obtained.

**Types of Studies Performed**

1. **Right Ventricular and Right Atrial Stimulation at Identical Rates**

Nineteen cases were studied. In 14 cases the right ventricle was stimulated first. After a series of two to three control measurements the right atrium or the right ventricle was stimulated at a progressive increase in rate up to a maximum of 180 per minute. Nine pairs of determinations were made for each chamber in the following order: the patient's own rate, 80, 100, 120, 140, 160, and 180 beats per minute. The artificial stimulation for any given rate lasted approximately 5 minutes, and measurements were obtained at the end of the period. In 12 cases the right atrium could not be stimulated at rates above 120 to 140 per minute because of non-conduction at the A-V junction.

The patients were divided into three groups: group 1, eight normal subjects; group 2, seven patients with compensated heart disease, and group 3, four patients in heart failure.

2. **Right Ventricular Stimulation at Various Sites**

Six patients were studied. Initially, two or three control measurements were obtained. The tip of the electrode catheter was then positioned in the outflow tract of the right ventricle. Several minutes elapsed before measurements were obtained to determine with certainty by using a fluoroscope whether the tip of the catheter had moved to another position in the ventricle. The pacemaker was then turned on to a rate of 100 per minute. Measurements were made at this stimulation site. Subsequently, the tip of the catheter was located in the midportion of the right ventricle, and another set of measurements was obtained. Finally, the tip of the catheter was positioned in the inflow tract of the right ventricle, and the same procedure repeated. The changes in the configuration of the QRS complex of the electrocardiogram demonstrated that in fact the site of stimulation had changed when the catheter was repositioned in the ventricle.

3. **Right Ventricular and Left Ventricular Stimulation at Identical Rates**

The tip of the electrode catheter was placed in the midportion of each chamber. Six cases were studied. In three cases the left ventricle was stimulated first, and in the remaining three cases the right ventricle was the first chamber to be paced. After a series of two to three control measurements the right or the left ventricle was stimulated at a progressive increase in rate (80, 100, 120, 140, 160, and 180 per minute) up to a maximum of 180 per minute. For each set of rates the stimulation lasted from 3 to 5 minutes and measurements were obtained at the end of the period.

**Results**

**Right Atrial and Right Ventricular PACING at Various Rates**

**Group 1. Normal Subjects**

Eight persons were studied. The mean control heart rate was 81 for right atrial pacing and 80 for right ventricular pacing. When the pacemaker was turned on with the rate of 80

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*Electronics for Medicine, North White Plains, New York.

*Complete data in rough form may be requested from Dr. Benchimol.
per minute, there was an initial fall in cardiac index, stroke index, mean arterial pressure, ventricular power, tension-time index, ejection time, stroke power, and stroke work for both atrial and ventricular stimulation. With an increase in rate of pacing there was a mild stepwise increase in cardiac index, tension-time index, dp/dt of arterial pressure with a stepwise fall in stroke index, peripheral resistance, ejection time, stroke power, stroke work, and systolic ejection rate (figs. 1 and 2). At a pacemaker rate of 120 per minute the mean difference between right atrial and right ventricular pacing for the cardiac index, stroke index, mean arterial pressure, ventricular power, and ejection time was $0.14 \pm 0.53$ L/min/m², $3 \pm 5$ ml/beat/m², $2 \pm 12$ mm Hg, $0.6 \pm 1$ kg-m/min/m² and $36 \pm 32^*$ msec, respectively. These figures were not statistically significant as P values were not less than 0.05. Further increase in rate of atrial and ventricular pacing to a maximum rate of 180 per minute resulted in a fall in cardiac index and ventricular power and a rise in peripheral resistance while stroke index, ejection time, stroke power, stroke work, and systolic ejection

*The number to the right of ± sign represents 1 standard deviation.
initial fall in cardiac index, stroke index, ventricular power, and dp/dt of arterial pressure for both right atrial and right ventricular stimulation. As the rate of stimulation increased, cardiac index, tension-time index, and peripheral resistance rose up to rates of 120 to 140 per minute for both right atrial and right ventricular pacing. Further increase in rate of stimulation resulted in a progressive fall in cardiac index, stroke index, ejection time, stroke power, stroke work, and systolic ejection rate for both right atrial and right ventricular pacing (figs. 3 and 4). Mean arterial pressure, ventricular power, and rate continued to fall. At the point of maximal stimulation cardiac index, stroke index, peripheral resistance, ventricular power, tension-time index, dp/dt of arterial pressure, stroke power, stroke work, and systolic ejection rate were slightly higher for right atrial pacing as compared with right ventricular pacing. However, there was considerable variation within the group, and the difference was not statistically significant.

**Group 2. Compensated Heart Disease**

Seven patients were studied. The mean control cardiac index was 2.94 L/min/m² prior to right atrial and right ventricular pacing. When the pacemaker was turned on at a rate of 80 per minute, there was an

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**Figure 3**

Mean cardiac index (C.I.), stroke index (S.I.), mean femoral artery pressure (F.A. mean), peripheral resistance (P. Res.), and ventricular power (V.P.) in group 2 patients with compensated heart disease, during stimulation of the right atrium (RA) and right ventricle (RV) at various rates. Pac = pacemaker. C = control.

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**Figure 4**

Mean tension-time index (T.T.I.), dp/dt of femoral artery pressure, ejection time (E.T.), stroke power (S.P.), stroke work (S.W.), and systolic ejection rate (S.E.R.) in group 2 patients during artificial stimulation of the right atrium (RA) and right ventricle (RV) at various rates. Pac = pacemaker. C = control.
dp/dt did not change significantly. For any given rate of stimulation, cardiac index, stroke index, mean arterial pressure, ventricular power, tension-time index, dp/dt, ejection time, stroke power, stroke work, and systolic ejection rate were generally slightly higher for right atrial pacing as compared with right ventricular pacing, and this was more evident at higher rates of pacing. At a pacemaker rate of 100 per minute the mean difference between right atrial and right ventricular pacing for cardiac index, stroke index, mean arterial pressure, ventricular power, and ejection time was 0.47 ± 0.62 L/min/m² 4 ± 6 ml/beat/m² 9 ± 9 mm Hg, 1.6 ± 1.2 kg-m/min/m² and 25 ± 27 msec, respectively. The figures were not statistically significant. The changes in ventricular power were significant to the 0.001 level and the ejection time to 0.01 with higher figures for right atrial pacing.

Group 3. Heart Failure

Four cases were studied. The mean control cardiac index was below the limits of normal being 2.33 L/min/m² for right atrial

Figure 5

Electrocardiogram (ECG), phonocardiogram (PCG) at the mitral area (MA), right ventricular (RV) and femoral artery (FA) pressures in one subject of group 3 before and during pacing of the right atrium and right ventricle. Note an increase in right ventricular systolic and end-diastolic pressures during ventricular pacing. The femoral artery pressure is higher during atrial pacing.
Mean cardiac index (C.I.), stroke index (S.I.), mean femoral artery pressure (F.A. mean), peripheral resistance (P. Res.) and ventricular power (V.P.) in a group of patients with heart failure (group 3) during artificial stimulation of the right atrium (RA) and right ventricle (RV) at various rates. Pac = pacemaker. C = control.

Figure 6
Mean cardiac index (C.I.), stroke index (S.I.), mean femoral artery pressure (F.A. mean), peripheral resistance (P. Res.) and ventricular power (V.P.) in a group of patients with heart failure (group 3) during artificial stimulation of the right atrium (RA) and right ventricle (RV) at various rates. Pac = pacemaker. C = control.

Figure 7
Mean tension-time index (T.T.I.), ejection time (E.T.), stroke power (S.P.), stroke work (S.W.) and systolic ejection rate (S.E.R.) in patients in heart failure (group 3) during artificial stimulation of the right atrium (RA) and right ventricle (RV) at various rates. Pac = pacemaker. C = control.

pacemaker rate of 100 per minute the mean difference between right atrial and right ventricular pacing for cardiac index, stroke index, mean arterial pressure, ventricular power, and ejection time was 0.49 ± 1.5 L/min/m² 4 ± 12 ml/beat/m² 12 ± 20 mm Hg, 1.2 ± 2.6 kg-m/min/m² and 18 ± 46 msec, respectively. These figures were not statistically significant as P values were not less than 0.05.

A-V Junction Conduction
Twelve patients developed varying degrees of nonconduction at the A-V junction at rates of atrial pacing above 120 to 140 per minute. In these patients an increase in rate of pacing resulted in a progressive increase in the P-R interval up to a point at which the refractory period of the A-V junction was reached re-
resulting in nonconduction with consequent reduction in ventricular response. Further increase in rate of pacing beyond the refractory period of the A-V junction resulted in a greater degree of nonconduction with further reduction in the ventricular response. An increase in the current of the pacemaker signal had no effect on the characteristics of this response. In three patients intravenous administration of 1 mg of atropine sulfate given at the rates which produced nonconduction at the A-V junction resulted in abolition of the block with return of 1:1 conduction. Under the influence of this drug the rate of atrial pacing could then be increased up to a range of 160 to 180 per minute with 1:1 conduction. The effect of this drug on A-V junction conduction was apparent within 1 minute after its administration. A systematic study of this problem and the effect of various drugs on this type of block is presently being made in our laboratory.

**Right Ventricular Pacing Sites**

Six persons were studied. Three were normal, two had compensated coronary artery disease, and one had congenital complete heart block. Since no differences were found between patients with heart disease and normal subjects, the results will not be described separately.

During this study the outflow tract of the right ventricle was paced initially, and this was followed by stimulation of the midportion of the right ventricle and lastly by stimulation of the inflow tract of the right ventricle. The rate of stimulation was fixed at 100 per minute.

No significant differences were found for the three pacing sites for cardiac index, stroke index, systolic, diastolic, and mean arterial pressures, dp/dt of arterial pressure, right ventricular pressure, ejection time, tension-time index, stroke power, mean systolic ejection rate, and stroke work (fig. 8). Cardiac index, ventricular power, stroke power, and systolic ejection rate were slightly lower for the group during mid-right ventricular pacing, but this was not statistically significant. The mean difference between pacing the outflow tract and the midportion of the right ventricle for cardiac index, stroke index, mean arterial pressure, ventricular power, and ejection time was 0.06 ± 0.3 L/min/m², 2 ± 2.5 ml/beat/m², 2 ± 4 mm Hg, 0.29 ± 0.5 kg·m/min/m², and 3 ± 3 msec, respectively. The figures were not statistically significant as P values were not less than 0.05.

The mean difference between pacing the midportion and the inflow tract of the right ventricle for cardiac index, stroke index, mean arterial pressure, ventricular power, and ejection time was 0.19 ± 0.13 L/min/m², 2 ± 1 ml/beat/m², 1 ± 5 mm Hg, 0.36 ± 0.5 kg·m/min/m², and 1 ± 3 msec, respectively. The figures were not statistically significant as P values were not less than 0.05.

**Right Ventricular Versus Left Ventricular Pacing**

Six persons were studied. Three were nor-

*Mean cardiac index (C.I.), stroke index (S.I.), mean femoral artery pressure (F.A. mean), dp/dt of femoral artery pressure, peripheral resistance (P. Res.), ventricular power (V.P.), ejection time (E.T.) and tension-time index (T.T.I.) in six patients during stimulation of the outflow tract (out), midportion (mid) and inflow tract (inf.) of the right ventricle at a fixed heart rate of 100 per minute.*
mal, two had compensated coronary artery disease, and one had compensated isolated mitral stenosis. The left ventricle was stimulated first in three cases, and the right ventricle was stimulated first in the remaining three. The rate of stimulation ranged from 80 to 180 per minute. The changes observed for cardiac index, stroke index, and other parameters of cardiac function as a function of heart rate were essentially the same as those described for right atrial and right ventricular pacing in patients with compensated heart disease (group 2 of study 1). For any given rate of right ventricular and left ventricular stimulation, there were no significant differences for cardiac index, stroke index, arterial pressures, right and left ventricular pressures, dp/dt,

![Graph](image-url)

**Figure 9**
Mean cardiac index (C.I.), stroke index (S.I.), femoral artery pressure (F.A. mean), peripheral resistance (P. Res.), and ventricular power (V.P.) in six subjects during artificial stimulation of the right (RV) and left (LV) ventricles at various rates. Pac = pacemaker. C = control.

![Graph](image-url)

**Figure 10**
Mean tension-time index (T.T.I.), dp/dt of femoral artery pressure, ejection time (E.T.), stroke power (S.P.), stroke work (S.W.), and systolic ejection rate (S.E.R.) during artificial stimulation of the right (RV) and left (LV) ventricles at various rates. Pac = pacemaker. C = control.

At a rate of 140 per minute the mean difference between right ventricular and left ventricular pacing for cardiac index, stroke index, mean arterial pressure, ventricular power, and ejection time was $0.10 \pm 0.3 \text{ L/min/m}^2$, $0.7 \pm 2 \text{ ml/beat/m}^2$, $3 \pm 3 \text{ mm Hg}$, $0.1 \pm 0.7 \text{ kg-m/min/m}^2$, and $15 \pm 27 \text{ msec}$, respectively. These figures were not statistically significant as $P$ values were not less than 0.05.
Discussion

Experimental studies in dogs by Wiggers\textsuperscript{15} demonstrated that pacemaking sites significantly influence cardiac performance. He showed that ectopic beats produced by ventricular stimulation resulted in less effective contraction by the ventricle from which the ectopic beats had originated as compared with those from the opposite ventricle. Subsequent studies by Corday and Irving\textsuperscript{16} in dogs showed that the site of an ectopic ventricular focus is an important determinant of cardiac output, coronary blood flow, and arterial pressure at rates below 160 per minute. More recently, Lister and associates\textsuperscript{17} provided further data substantiating the fact that the site of artificial ventricular stimulation from the epicardium significantly alters cardiac performance in dogs.

Our observations on patients with normal and abnormal hearts with \textit{endocardial} right ventricular and left ventricular pacing and at various sites in the right ventricle do not support the previous observations on dogs. No significant differences were found at various pacing sites in the right ventricle or between the left ventricle and the right ventricle. Therefore, we are in agreement with the work of Fletcher,\textsuperscript{10} Levy,\textsuperscript{17} Starzl\textsuperscript{18} and their associates and, by Williams-Olsson and Anderson,\textsuperscript{19} who demonstrated that sites of artificial pacing do not significantly alter cardiac performance. At any given rate of stimulation up to a maximum of 180 per minute, right ventricular and left ventricular stimulation results in similar changes in cardiac output, stroke volume, mean arterial pressure, ventricular power, and other parameters of ventricular performance in both normal and abnormal hearts. Furthermore, our data, at a fixed rate of 100 per minute at various pacing sites in the right ventricle provide further documentation that the site of an ectopic focus does not significantly affect ventricular performance in man, either in normal or abnormal hearts. These data have practical application because of an increase in the number of permanent pacemakers which are being used for treatment of complete heart block. The wires of these pacemakers are most commonly implanted in the left ventricular wall. Our data suggest that these wires may be implanted in the right ventricle if necessary since the hemodynamics of these two pacing sites are nearly identical.

Cardiac output in the group of normal subjects increased only slightly with an increase in rate. For a wide range of rate cardiac output remained relatively stable but fell at rates above 150 per minute. However, in the group of patients with heart disease with or without failure in whom cardiac output was lower in the control figures, it rose with increase in rate up to a range between 120 and 150 per minute. A further increase in rate resulted in a marked fall in cardiac output. These data confirm Braunwald and associates' observations\textsuperscript{20} who also did not detect significant changes in cardiac output during a wide range of atrial pacing. The mechanisms involved in these two types of responses of the normal and abnormal heart are not known. It is conceivable that a normal heart may accommodate large variations in stroke volume at the expense of changes in stroke work and ventricular power whereas in the diseased heart cardiac output rises with an increase in rate because, to a great extent, there are considerable limitations on the variation of the stroke volume. Therefore, in these patients, the increase in heart rate is the predominant mechanism for the increase in cardiac output. The mechanisms of the fall in cardiac output at faster rates have been described by Wiggers and Katz,\textsuperscript{21} Wiggers,\textsuperscript{22} Nakano,\textsuperscript{23} and Wegria,\textsuperscript{24} and Miller\textsuperscript{25} and their colleagues, and are most likely related to a decrease in diastolic filling time which results in a decrease in coronary blood flow and myocardial hypoxia.

It has long been suggested that the atria significantly contribute to ventricular filling.\textsuperscript{26–30} Most of the earlier studies\textsuperscript{26–28, 31} indicated that the contribution of the atria to cardiac output ranged from zero to as much as 30%.

The importance of atrial systole to cardiac function in man has only recently been the object of systematic study.\textsuperscript{32–35} In previous
observations\textsuperscript{33} on patients with complete heart block we have shown that a properly timed atrial contraction (P-R interval of 1 to 300 msec) results in an increase in ejection time (13\%), isometric contraction time (6\%), systemic arterial systolic pressure (15\%), dp/dt of arterial pressure (14\%), and tension-time index (33\%) as compared with figures obtained during a time when atrial systole occurs during ventricular systole. Furthermore, our studies indicated that the contribution of atrial contraction to the increase in these parameters occurs at a wide range of ventricular rate (20 to 125 per minute). However, no measurement of cardiac output was obtained during that study and the question was not answered as to whether or not atrial contraction significantly improves cardiac output in man. Gilmore and associates\textsuperscript{36} have shown in dogs that the left ventricle produces less stroke work than atrial pacing does for any given left ventricular end-diastolic pressure for ventricular pacing.

The present study indicates that in normal human hearts cardiac output, stroke volume, ventricular power, and ejection time are only slightly higher during atrial pacing than during ventricular pacing for any given rate up to a maximum of 180 per minute. However, in the group with compensated heart disease and in the group with heart failure, the contribution of atrial systole appears to be of greater significance. This difference appears to be slightly higher at faster rates of pacing.

More recently, Samet and associates\textsuperscript{35} indicated that synchronous atrial and ventricular pacing results in slightly higher cardiac output than asynchronous pacing does. However, this difference is relatively small and variable. Data obtained from patients with atrial fibrillation and after conversion to regular sinus rhythm have shown variable improvement in cardiac function.\textsuperscript{37-39} Our own data\textsuperscript{39} did not demonstrate any significant increase in cardiac output at rest or during exercise after conversion to sinus rhythm.

Recently, Braunwald and associates\textsuperscript{40} emphasized that atrial contraction does not significantly elevate left ventricular end-diastolic pressure in the presence of ventricular dilatation. Burchell\textsuperscript{41} also stated that atrial contraction does not appear to be an important regulatory mechanism of cardiac output. It does become apparent that under certain circumstances atrial systole may become an important factor to the cardiac function. However, this contribution is highly variable and to a great extent unpredictable. Nevertheless, this contribution to cardiac output is relatively small and in the majority of cases the human heart may function at a wide range of work loads without the contribution of a coordinated atrial contraction.

Summary and Conclusions

Artificial stimulation of the right atrium and the right ventricle at rates between 80 and 180 per minute was performed in eight normal subjects, in seven patients with compensated heart disease, and in four patients with heart failure. For any given rate of atrial or ventricular stimulation there were no significant differences for cardiac index, stroke index, mean arterial pressure, ventricular power, and ejection time in normal subjects. In patients with compensated heart disease there was a slight tendency for higher figures for these parameters during atrial pacing, but these differences were not statistically significant.

Stimulation of the right and left ventricles at equivalent rates in the range of 80 to 180 per minute did not show any statistically significant changes for cardiac index, stroke index, mean arterial pressure, stroke work and power, ventricular power, ejection time, tension-time index, and peripheral resistance.

Stimulation of the outflow tract, the midportion and the inflow tract of the right ventricle at a fixed rate of 100 per minute did not show any significant changes in cardiac index, stroke index, mean arterial pressure, and other related parameters.

It is concluded that pacing sites do not significantly influence cardiac dynamics in unanesthetized man. Furthermore, the contribution of atrial systole to the cardiac function is minimal in normal subjects but slightly greater in patients with heart disease.
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