Arrhythmic and Antiarrhythmic Effects of Sodium, Potassium, and Calcium Salts and of Glucose Injected into Coronary Arteries of Infarcted and Normal Hearts

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Substances that have not exhibited properties as excitants of ectopic activity by intracoronary injection into normal hearts often produce rapid ectopic activity and sometimes ventricular fibrillation upon slow injection through the vascular bed of an infarcted area, particularly upon the first one or two injections. Sodium lactate has reduced and stopped ectopic activity for brief periods. Excesses of both potassium and calcium have increased ectopic activity in infarction and produced it in normal hearts. No antagonism between these two ions, with respect to ectopic activity, has been demonstrated.

Observations which suggest strongly that local increase in potassium concentration is a significant factor in the excitation of ectopic impulses in regional ventricular ischemia and infarction have been reported briefly.\(^1\) Injection of potassium chloride into a coronary artery of a normal dog heart with continuing circulation produces ectopic activity. Occlusion of a coronary artery causes potassium to be liberated in enough quantities in the ischemic region to elevate significantly potassium concentration in coronary venous blood from that area. The local potassium concentration curve shows 2 elevations. The first begins promptly after ligation, reaches an early maximum within 5 to 10 min. and then declines toward the control, but usually stabilizes somewhat above it by the end of 30 min. This intermediate level is maintained for about 3 hours, after which a delayed rise begins. The maximal local potassium concentration, usually between 1\(\frac{1}{2}\) and 2 times the control, is reached within the first 24 hours of occlusion and declines irregularly during the following hours and days. Tissue analysis shows that sodium is gained in the dying muscle as potassium is lost.\(^2\)

During the first few hours of occlusion, while the local changes are occurring, the systemic venous blood shows no corresponding changes in potassium content. The local changes therefore signify varying potassium concentration differences across the boundary where ischemic muscle joins muscle with normal blood supply. After 24 hours the systemic blood usually has an increased potassium concentration also, approximating the level in the coronary vein in the ischemic region. Excitation by difference in potassium concentration across a boundary could be operative during the early periods of elevation, accounting, hypothetically, for the ectopic excitation of the first 10 min. of occlusion and for the first several hours of the delayed ectopic activity, the onset of which is 4\(\frac{1}{2}\), 5 or 6 hours later. The period of lack of ectopic activity between these phases coincides in time with the period of reduction from the first elevation of potassium concentration and with the first hour or two of the subsequent gradual rise which begins about 3 hours after occlusion. Some excitatory factor, or factors other than potassium difference across a boundary, appears necessary.
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to carry on the arrhythmia after 24 hours. If freer movement of ions across the membranes of partially ischemic living cells in the boundary is a factor in ectopic excitation, this could account for persistence of ectopic activity far beyond the time for establishment of electrolyte equilibrium between nonviable infarction muscle and extracellular fluid.

In the presence of autolyzing tissues the creation or liberation of multiple substances (including nonelectrolytes), which could excite a hypersensitive, injured border zone, appears possible. Evidence that multiple excitatory factors do operate in different degrees in infarction-induced arrhythmia is found in experiments with procaine amide.

In some dogs having very high frequency ventricular tachycardia (250 to 300/min.) procaine amide in moderate dosage reduced the ectopic frequency to a relatively low range (20 to 80/min.) within a short time, but multiple additional doses failed to bring about consistent, further reduction or to eliminate ectopic activity. In some other animals with low ectopic rates (20 to 90/min.) before administration, procaine amide produced little change in the ectopic rate or the pattern of fluctuations. In still other animals it completely suppressed ectopic activity for periods of time that varied with dosage.

The interpretation of these differences is that procaine amide in effect antagonizes one excitatory factor that is operative in some cases of infarction ventricular tachycardia, and fails to antagonize some other factor or factors. In the production of these arrhythmias, the factors both sensitive and insensitive to procaine amide are additive, and vary greatly in relative proportions from almost complete control by one to almost complete control by another or others.

To obtain further information upon the role of electrolytes in the excitation and suppression of ectopic impulses, a number of substances such as sodium lactate, potassium chloride, calcium chloride, Locke's solution and glucose have been administered, principally by intracoronary injection of animals with infarction arrhythmias and with normal hearts.

METHODS

Myocardial infarction was produced in the hearts of dogs weighing 9 to 12 Kg. by the method used in previous studies. With the use of pentobarbital sodium anesthesia, 30 mg./Kg., artificial respiration and aseptic surgical methods, the heart was exposed via an incision in the fourth intercostal space on the left side. The anterior descending artery was dissected free from adjacent structures and enough to allow the passage of ligatures for occlusion at the diastolic level of the free edge of the left atrial appendage. The artery was occluded in two stages. The first ligature was tied snugly but not tightly around the artery together with a 20 gage hypodermic needle, and the needle was withdrawn immediately. After 30 min. of partial occlusion the second ligature was tied tightly to complete the closure.

A polyethylene catheter was then inserted just distal to the ligature and tied in place for the purpose of later injections through the coronary bed of the ischemic muscle. A silver wire in the catheter kept it open until needed. The wound was closed and the dog was given careful postoperative attention, and fluid and morphine when needed. On the following day a number of control electrocardiograms were made, the silver wire was removed, and the effects of test substances were recorded.

In control experiments injections and infusions into nonoccluded, anterior descending arteries were made. For this purpose a catheter was inserted into the left coronary orifice in the aorta and passed into the artery a distance of about 2 cm., or just beyond the distal edge of the left atrial appendage. The catheter was directed to the orifice by use of a tubular metal guide which entered the right common carotid artery in the lower part of the neck and was carefully passed into the root of the aorta and aligned with the orifice. The heart was exposed for palpation, and for observation through an incision in the left fourth intercostal space. In using this method, the artery remains open at all times. A catheter, with the end segment smaller than the lumen of the artery, was used. Mepesulfate (Roche) was injected to prevent coagulation in the catheter. The initial dose, 10 mg./Kg., was followed by additions of 5 mg./Kg. hourly.

RESULTS

Intracoronary Injections Distal to Occlusion in Infarcted Hearts. Injections on the first postocclusion day of a variety of substances through the portion of the coronary

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system that perfuses the region of infarction has produced results which in some cases were not to be anticipated from previous knowledge of their biological actions.

**Sodium Lactate.** The slow intracoronary injection of sodium lactate solution in 6 dogs increased ectopic activity in 3 of them and produced ventricular fibrillation in 2. In 3 others similar administration has had the opposite effect, i.e. it has reduced and stopped ectopic activity for significant periods. The production of increased ectopic activity and ventricular fibrillation are illustrated in figure 1. Twenty-four hours after occlusion, after a number of control records had been made, 10 ml. of 1/6 M sodium lactate was injected in 3 min. via catheter in the occluded artery. Prior to injection the ectopic rate had been 150/min. and the impulses were heterotopic in origin. At the end of the injection the ectopic rate had increased to 230, and the impulses were from a single focus. After a wait of another minute, during which there was little change, an additional 15 ml. was injected in 3 min. About 1 min. later the action changed to a fast flutter, and changed again after 40 sec. to ventricular fibrillation. In the other case of ventricular fibrillation after sodium lactate the injection of 25 ml. in 6 min. slightly reduced the ectopic rate from 205 to 195/min. and the pattern indicated a shift to a higher ectopic pacemaker. This was followed 2 min. later by a new increase in ectopic activity which led to ventricular fibrillation 4 min. after injection. In this case there is a suggestion of diphasic effects: reduction of ectopic activity followed by increase.

The reduction and stoppage of ectopic activity by intracoronary injection of 1/6 M sodium lactate is illustrated in figure 2. Beginning 26 hours after occlusion, 4 control records at 15 min. intervals were made. Each of them showed ventricular tachycardia,
heterotopic in origin, at a rate of about 160/min. At the end of an intracoronary injection of 15 ml. in 7 min., regular sinus rhythm at a rate of 150 was recorded. Some ectopic activity had recurred 8 min. later, and after 13 min. there was again a completely ectopic ventricular rhythm. The lowest record shows another remission after injection of only 5 ml. in 2 min. but this remission was of short duration, being superseded by a return of ventricular tachycardia (not illustrated). Administration of 10 ml. in 5 min. restored the sinus rhythm. This lasted about 10 min., after which there was another recurrence of ventricular tachycardia. A final dose of 10 ml. in 3 min. brought another remission which lasted much longer, a gradual return of ectopic beats beginning after 50 min.

The experiment described and 2 others in which repeated and unquestionable remissions were produced, have demonstrated that sodium lactate by the intracoronary route can neutralize the excitatory factors that exist in infarction in some animals. The failure to produce significant inhibitory or excitatory results in 2 animals is unexplained. Each catheter was proved to be fixed in the artery, but the solution may have channeled through special paths without distribution to active or potential trigger zones.

Sodium lactate, 0.5 M or 1 M, intravenously administered in 5 dogs, yielded significant remissions of short duration in 3 of them. Re-
Repeated injections were made in each animal. The most effective doses were of 1 M solution, 40 ml. in 1 min. Complete remission was fleeting, beginning during injections and lasting less than 1 min. after an injection was completed. Longer durations of reduction of ectopic rate by one half were observed after 4 or 5 repeated injections in each of the animals in which remissions were recorded.

Excess Calcium. As CaCl₂, excess calcium, added to Locke's solution to make calcium concentrations 2, 4, 6, and 8 times normal, has been administered via coronary catheter in repeated trials to 4 dogs with infarction arrhythmia. Ectopic activity was increased in every animal following certain injections. The sensitivity usually appeared greater than normal upon first or second injections, but this may have been due to nonspecific factors, since in subsequent injections the threshold dose tended to increase toward that of normal hearts: calcium 8 times normal, 20 ml. in 1 minute (as will be described). Following some injections there were reductions of ectopic activity, including periods of normal complexes that were not recorded at other times. These reductions followed increases in each case, with one exception. Increases were more readily produced than decreases. These tests were made in the presence of mixed normal and ectopic complexes of moderate frequency. The results of two injections are illustrated in figure 3.

The temptation to assume that the few reductions observed resulted from a stabilizing effect of calcium is discouraged by the considerations that this result was undependable, that when it occurred it tended to follow a
period of increased ectopic frequency, and that reduction was never observed to result from the intravenous injections of calcium chloride which will be described.

In 4 other dogs, calcium chloride (1.6 per cent) was administered intravenously in increasing doses, up to total amounts that were calculated to contain 3 to 4 times the quantity of calcium normally present in the dog’s blood plasma. No reduction of ectopic activity was recorded, but a slight increase was observed in some trials, e.g. in one experiment from an average frequency of 195 to 215/min.

**Excess Potassium.** Effective doses in the form of potassium chloride enriched Locke’s solution or blood, injected into a coronary artery on the first or second postocclusion day, has consistently produced ectopic activity if none existed before, and increased the frequency if moderate level or low level activity was present. Such injections have never decreased the ectopic rate. Injections made during high-frequency ventricular tachycardia have had less effect than in the presence of lower frequencies. The ectopic threshold to excess potassium shows wider variations in these experiments than in noninfarcted hearts. The threshold dose may be within the normal range, as in the trial shown in figure 4, but often it is distinctly higher than in the normal heart. Injections of excess potassium into the infarcted heart have proved to be prone to produce less ventricular fibrillation than in the normal heart, even when ectopic frequency is markedly increased. The tendency to produce ectopic impulses for long periods (up to 20 or 30 min. after injection) or to produce delayed ectopic activity is greater in infarcted hearts than in normal hearts. Prolonged excitation and delayed excitation (beyond 2 or 3 min.) have not been observed in any of the other substances used for intracoronary injections.

**Glucose.** A 5 per cent solution with 1/2 unit of insulin/Gm. of glucose was administered to 9 dogs by infusions and injections via catheter to test effects upon infarction arrhythmia. These tests were prompted by the hypothesis that glucose insulin treatment might cause potassium to be absorbed into the cells, ectopic activity thereby being reduced. Some irregular declines in activity were recorded, but they cannot yet be regarded as significant. Increases in ectopic activity early in the testing were recorded in 5 dogs and ventricular fibrillation was induced in 3.

**Locke’s Solution.** Doses of 3 or 5 ml. were injected through the catheter into the occluded artery at the beginning of numerous tests to insure patency of the catheter and artery before administration of test solutions. The Locke’s solution had notably increased
ectopic activity following some of these in-
jecions. Results similar to that shown in
figure 1 following the first 10 ml. of sodium
lactate injection have been recorded after
Locke’s solution, but no ventricular fibrilla-
tions have been precipitated. It has not been
injected in quantities as great as those used
in tests of sodium lactate and glucose.

Results in Normal Hearts

Intracoronary injections into normal hearts
of 6 dogs were made with each of the solu-
tions which were used in intracoronary tests,
to determine the effects upon ectopic activity
in myocardial infarction. Representative
effects in these uninjured hearts are shown in
figures 5 and 6. Each horizontal line of 3
records contains a segment taken just before
injection, another taken during the last 4 sec.
of injection, and a third segment taken 30
sec. after the injection was complete.

Sodium Lactate 1/6 M. Twenty ml. in-
jected in 1 min. prolonged the Q-T segment
and changed the form and direction of T.
No ectopic beats and no change in rhythm
resulted. In the different dogs the forms of
the T waves differed in the control records,
and the changes in direction with sodium lac-
tate changed also. The prolongation of Q-T
was consistent, being recorded with each in-
tracoronary injection in every dog.

Excess Calcium. Injections of Locke’s so-
lution (not illustrated) and Locke’s solution
with 2 and 4 times the standard concentration
of calcium chloride produced no ectopic beats,
made no changes in duration of Q-T, and no
changes in rhythm. All injections were of 20
ml. in 1 min. Some changes in the form of
T wave were evident.

An eightfold excess of calcium chloride in
Locke’s solution, 20 ml. in 1 min., produced
ectopic impulses. This was about the thresh-
old dose for ectopic beats. The response var-
ied in the different animals from a single
ectopic complex, as illustrated in figure 5, to
an irregular succession totalling about 20.

Potassium Chloride Excess. Figure 6 illus-
trates the effects of potassium chloride three
times the normal in Locke’s solution, 10 ml.
in 20 sec. Such a solution produced ectopic
beats during or immediately following its in-
jection. In some trials, including the one re-
produced, delayed ectopic activity has oc-
curred also. A reasonably constant threshold
dose of potassium chloride for the imme-
-diate response can be determined in many animals.

Delayed ectopic activity, having its onset
or definite increase 5 to 20 min. after intra-
coronary injection of effective doses of potas-
sium chloride, is an inconstant phenomenon.
It has occurred more frequently in experi-
ments in which the artery had been occluded
for a few minutes during preparation (carot-
id coronary external loop) than in experi-
ments without occlusion (small catheter via orifice). From injections via the orifice cath-
eter, delayed reactions appear more probable
in late trials in an animal than in the first
one or two.

Glucose 5 Per Cent. Injections of 20 ml.
in 1 min. (not illustrated) produced some
changes in the form of T wave, but no ectopic
beats and no change in rhythm.

Intracoronary Injections of Excess Potas-
sium and Calcium Simultaneously into Nor-
mal Hearts. On 9 dogs experiments were per-
fomed for the purpose of testing the often
suggested hypothesis that, if excess potassium
ions induce ectopic impulses, an equivalent
excess of calcium ions should prevent them.
Six of the experiments yielded data which are
regarded as valid; in the other 3 no combina-
tion doses were administered because of the
unsuitable condition of the preparations. The
results of the potassium-calcium combination
experiments are represented by the selected
data in table 1.

In each experiment a dose of potassium-
calcium 1 per cent added to Locke’s solution,
sufficient to total 10 ml. which repeatedly pro-
duced a few ectopic beats, preferably without
ventricular fibrillation, was determined. Then
an equal dose was again administered, togeth-
er with a chemically equivalent quantity of
calcium ions as calcium chloride added in the
same syringe, again with Locke’s solution to
total 10 ml. The production of ectopic beats
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TABLE 1.—Ectopic Impulse-Inducing Effects of Excess Potassium and of Chemically Equivalent Excesses of Potassium and Calcium Chlorides in Locke’s Solution by Intracoronary Injection

<table>
<thead>
<tr>
<th>Time</th>
<th>Salts</th>
<th>Dose* (mg./Kg.)</th>
<th>Immediate ectopics</th>
<th>Ventricular fibrillation</th>
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<td>KCl</td>
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<td>No</td>
</tr>
<tr>
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<td>(KCl</td>
<td>1.6</td>
<td>10</td>
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</tr>
<tr>
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<td>No</td>
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</tr>
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<td>6</td>
<td>No</td>
</tr>
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</tr>
<tr>
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</tr>
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<td>KCl</td>
<td>2.0</td>
<td>17</td>
<td>No</td>
</tr>
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</table>

*Since the threshold dose varies more widely between animals than between repeated trials in the same animal, each animal provides its own control observations. Stated doses of these salts in isotonic solutions were measured, and sufficient Locke’s solution added to total 10 ml. Duration of each injection was 20 sec. (variation 19-21). Threshold doses of added KCl correspond to injected concentrations of K, 3 to 5 X normal.

DISCUSSION

The production of excitatory effects by non-excitatory fluids, upon their injection through the vascular bed of an area of infarction, has raised questions as to how it occurs. It is conceivable that the injected fluid mobilizes stagnant extravasated fluid and relatively stagnant quantities of fluid in vessels whose fluid has been somewhat isolated from the circulation, possibly by clots, and that these fluids contain substances with excitatory properties. The concept that the injected fluid moves excitatory material to contact live tissue or more sensitive trigger zones, and thereby increases ectopic activity, may be supported (see fig. 1) by the observation that, after the initial injection of a small volume of fluid has produced an increase in ectopic excitation, an additional injection results in an intensification of this excitation. There is some relation to volume. It has been shown, however, that such results are limited. After 2 or 3 injections, further injections do not have excitatory effects, and may have little effect of any kind, but the previous level of ectopic activity continues with little change. This could mean that no further excitatory material is to be mobilized in the regions reached by the fluid, and that isolated ectopic foci are continuing undisturbed by their previous rate of activity.

It is highly improbable that pressure of the injected fluid is a factor in the excitations
observed, since each injection is made slowly (e.g., 10 ml. in 3 min.) and the principal effect often is manifest after it is completed.

The observation has again been confirmed that excess potassium has excitatory effects when injected into a coronary ramus that perfuses a region bounded by normal tissue with undisturbed circulation. The excitatory effect of injected potassium is demonstrable in infarction also, and is not limited to the first injections, as is the case with the substances that are not excitatory to uninfarcted hearts.

The finding that sodium lactate exhibited antiarrhythmic effects in a significant proportion of experiments by both intracoronary and intravenous administration apparently indicates that an excess of sodium ions has a stabilizing effect under the conditions of these experiments. Sodium lactate was the only test substance that prolonged the Q-T segment (closely correlated with the refractory period) upon intracoronary injection into normal hearts. Bellet and collaborators, on the basis of observations in arrhythmic patients, found that sodium lactate restored normal rhythm in a significant proportion of cases. Sodium chloride was less effective than lactate.4

Calcium ions in excess have exhibited effects that were opposite to the stabilizing influence observed in studies with isolated Purkinje fibers. Weidmann 5 reported that a fourfold increase in calcium concentration exhibited a number of properties similar to those of cocaine hydrochloride, quinidine sulfate and procaine amide, including suppression of spontaneous activity. In the infarction arrhythmia calcium excess has produced only capricious, unrepeatable reductions and remissions by intracoronary administration, and none at all by the intravenous route. On the contrary, excess calcium more reliably increases ectopic activity by the intracoronary and intravenous routes in the normal and infarcted preparations. Quinidine compounds6 and procaine amide3 are effective suppressors of ectopic activity in infarction.

The finding that calcium excess fails to antagonize the ectopic impulse-inducing effect of potassium excess upon intracoronary injection need not be surprising. Other instances of nonantagonism of calcium and potassium are well known. For example, Bishop 7 has shown that potassium chloride (1 per cent), applied to frog motor nerve fibers, depressed resting and action potentials and blocked conduction within 2 min., and that Ringer's solution, mixed with isotonic calcium chloride in equal proportions, did not act oppositely to potassium chloride but depressed both potentials to final block. Grundfest and co-workers8 found that a microinjection into giant axons of an excess of each of 8 cations, including potassium, sodium, calcium and magnesium, caused decrease in spike amplitude and block. In infarction magnesium is a potent inhibitor of ectopic impulses; 8 sodium has shown weaker inhibitory effect; potassium and calcium increase ectopic activity. It is clear that the parameters thus far measured in single Purkinje fiber and nerve fiber experiments have not accounted for the excitatory factors that exist at a boundary where infarcted heart tissue joins normal myocardium, nor for the factors that obtain when ejection excesses are injected into a coronary ramus. The presence of a boundary seems to create important differences.

**SUMMARY**

Observations supporting the hypothesis that locally liberated potassium is one of two or more factors that must exist in ectopic excitation in infarction are reviewed.

Substances that had exhibited no ectopic excitatory effect by intracoronary injection in uninfarcted hearts (1/6 M sodium lactate, 5 per cent glucose and Locke's solution) produced high-frequency ventricular tachycardia and ventricular fibrillation after slow injection through the occluded coronary artery in infarction. It is believed that the movement of products of necrosis with excitatory properties to responsive cells could account for the results.

Sodium lactate also produced antiarrhythmic effects. It was the only test substance that prolonged the Q-T interval of normal hearts.
Excesses of potassium chloride and of calcium chloride induced ectopic activity in both normal and infarcted hearts upon intracoronary injection. Calcium chloride also induced ectopic activity upon intravenous administration.

Calcium chloride added to an ectopic threshold dose of potassium chloride in equivalent concentration failed to prevent the production of ectopic beats in normal hearts in experiments with intracoronary injections.

Some effects of ions upon single Purkinje fiber and nerve fiber preparations are cited. Some important differences between those effects and others observed in heart experiments are believed to be attributable to the boundary between the injected coronary bed (normal and infarcted) and the adjacent muscle.

Summario in Interlingua
Es presentate, como introduction, un revisata de observationes que supporta le hypothese que kalium de liberation local es un de duo o plure factores que debe exister in excitation ectopic in casos de infarcimento.

Substantias que non exhibiva ulle effecto de excitation ectopic post lor injection intracoronari in cordes sin infarcto (i.e. lactato de natrimum de M/6, glucosa de 5 pro cento, e solution de Locke) se provava capase a producere tachycardia ventricular e fibrillation ventricular de alte frequentia post lor lente injection via le occcludite arteria coronari in cordes con infarcto. Es opinate que le movimento de productos necrotic con proprietates excitatorie una celular responsiva pote explicare iste resultatos.

Lactato de natrimum ha etiam producere effectos anti-arrhythmic. Ilo eseva le sol substantia testate que prolongava le intervallo Q-T in cordes normal.

Excessos de chloruro de kalium e de chloruro de calcium induceva activitate ectopic in cordes tanto normal como etiam infarite post injectiones intracoronari. Chloruro de calcium etiam induceva activitate ectopic post su administration intravenose.

Quando chloruro de calcium eseva addite a un dose a limine ectopic de chloruro de kalium in concentration equivalente, illo non preveniva le production de pulsos ectopic in cordes normal in experimentos con injectiones intracoronari.

Es mentionate certe effectos de ions super preparatos de fibras de Purkinje e fibras nervose individual. Le importanta differencias inter iste effectos e le effectos observate in experimentos con cordes es probablemente atribuibile al barriera inter le (normal o infarite) vasculatura que reciproc le injection e le musculo adjacent.

References
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