A variety of resuscitative measures are being employed currently both in experimental studies and in clinical episodes of cardiac arrest.\(^1\)\(^-\)\(^5\) While survival is certainly the best means of determining the efficacy of these resuscitative procedures, it would be valuable to be able to differentiate between those marginal techniques which are successful occasionally and others which might be responsible more regularly for a quick return to the prearrest cardiac status. Obviously, many normal dogs thrown into ventricular fibrillation by electric shock might be expected to recover from borderline resuscitative measures. These marginal efforts would probably be ineffective for patients with coronary artery disease who sustain ventricular fibrillation as a result of an acute occlusion. The observation that only a small number of these patients are restored suggests that the current resuscitative techniques may not be optimal.\(^4\)\(^-\)\(^5\)

In a previous report\(^6\) we studied some of the cardiorespiratory variables employed in resuscitation, using a mechanized pneumatic pump for external cardiac massage. The return of central nervous system function as well as the electrical activity and hemodynamic parameters of the heart were used in the evaluations. However, the appraisal of electrocardiographic changes was rather cursory, e.g., the time required for return to a nearly normal pattern being the main consideration. The electrocardiogram has become an invaluable tool in diagnosis of myocardial infarction, in evaluation of therapeutic measures, and in prognostication during the recovery period. Degrees of change in several components of the electrocardiogram have been used to a limited extent clinically, but more often experimentally, as a means of evaluating the amount of myocardial ischemia or damage.\(^5\)\(^-\)\(^10\) For example, giant R waves,\(^10\) ST segment deviation,\(^11\)\(^-\)\(^12\) T-wave changes,\(^13\) and the presence of Q waves\(^14\) have been used as criteria to differentiate mild from severe degrees of ischemia or necrosis. The quantitative features of these changes are not accepted universally as being meaningful; indeed, there are many who question the value of ever using the electrocardiogram as a measuring device. Despite these reservations, experimental evidence in general favors the view that certain changes are more regularly associated with severe degrees of ischemia or necrosis, while other changes are observed in less ominous circumstances.

The present study was undertaken to determine (1) whether the initial complex obtained after countershock can assist in immediate prognostication, (2) whether the electrocardiographic patterns during the first hour of recovery differ in animals which live as compared with those which die, and (3) whether in the surviving animals different degrees of ischemia can be recognized and quantitated. This last feature might serve to grade the value of the method of resuscitation used and help differentiate those measures which permit survival but are somewhat injurious from those techniques which not only permit survival but also return the animal to his prearrest status with a minimum of injury and in the shortest possible time.

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Methods

Forty-three mongrel dogs ranging in weight from 9 to 25 kg were anesthetized with pentobarbital sodium (25 mg/kg body wt iv), placed in the supine position on a V-shaped table, and arranged with the body supported laterally, under the piston of the previously described pump for external cardiac massage.* The femoral vessels were isolated for recording of arterial pressures and for administration of drugs during the recovery period. A tracheal tube was inserted.

The experimental design consisted of the following procedures. (1) Ventricular fibrillation was induced with a 120-volt shock (a-c) lasting 1 to 2 seconds. (2) After 1/2 minutes ventilation was begun with 100% oxygen, by tracheal tube, and 30 seconds later external cardiac compression was started. (3) Sodium bicarbonate was given intravenously at 15 and 29 minutes in order to combat the metabolic acidosis which results from the inadequate circulation. (4) After the designated duration of fibrillation (usually 30 minutes), defibrillation was attempted by delivering 450 volts to the chest wall for 0.25 second in single and multiple shocks, using the Kowen-hoven (a-c) defibrillator. Massage was continuous between countershocks until defibrillation was successful. When fibrillation was not terminated readily and a slow rate with low voltage was noted, epinephrine (1:1,000) was given by intracardiac injection. As reported by others,17-20 this drug appeared to improve the character of the fibrillation and after a short period of additional massage, the previously ineffective countershocks resulted in a conversion of the fibrillation to conducted beats.

During the recovery period it was frequently necessary to infuse norepinephrine in order to sustain an adequate arterial pressure. The amount of norepinephrine necessary and the duration of its administration affected the degree of recovery, as described elsewhere.9 Although it would have been preferable to study the electrical recovery following defibrillation without the use of drugs reputed to be arrhythmogenic, in practice (both experimentally and clinically) the need for some temporary support with vasopressor drugs is essential. In several early pilot experiments, survival could not be obtained without the use of norepinephrine even though, after a 30-minute period of ventricular fibrillation, countershock could convert the rhythm to a conducted beat. Occasionally when defibrillation was difficult and the duration of massage was prolonged thereby, additional sodium bicarbonate was given. Generally, one ampoule (44.5 mEq) was given during the experiment, but in the circumstances just mentioned twice this amount of alkali was used. Although we did not measure the serum pH levels in these experiments, similar doses of alkali in other resuscitation experiments never elevated pH to abnormal levels. Furthermore, when 22.5 mEq of sodium bicarbonate were infused into three normal animals at the rate used in the resuscitation experiments, no arrhythmias were produced, nor was the shape of the QRS complex altered in any way. Although this issue will be referred to again later in the text, these are some of the reasons why we feel that the use of bicarbonate, epinephrine, and norepinephrine does not invalidate the significance of the electrical changes which we observed in animals recovering from ventricular fibrillation, after having been massaged with different techniques.

The external cardiac massage was carried out for 30 minutes (in all but 10 of the animals) using the mechanized pump which permitted controlled changes in rate, duration of systole, and force of sternal compression. In seven dogs manual massage was performed for comparison. However, the results obtained in animals massaged for shorter times and by manual technique did not differ from those in the total group and thus are not described separately.

Control electrocardiograms (leads I, II, III, AVF, AVL, and AVF) were taken just before ventricular fibrillation was induced. Full tracings of this type were also recorded at 1, 24, and 48 hours after the initial electrically induced fibrillation. During the first 5-minute period after defibrillation, a nearly continuous recording of lead II was obtained; subsequently, 30 to 60-second strips of the same lead were taken about every 5 minutes, until 30 minutes after the induction of fibrillation.

In summary, for most animals the experiment consisted of a 30-minute period of ventricular fibrillation, during which viability was maintained by external cardiac massage and ventilation with pure oxygen, plus a 60-minute period initiated by external countershock, during which recovery was followed by observation and frequent recording of the electrocardiographic changes seen in lead II. Because of the need for disconnecting and reconnecting the electrical contacts for countershocking, and because of the necessity for continuing massage when an adequate blood pressure did not reappear immediately upon defibrillation, many of the tracings in the early recovery period contained both electrical and mechanical artifacts. However, with a little experience the interpretations of electrocardiograms in the presence of these artifacts did not present serious difficulties.

* Westinghouse Corporation, Baltimore, Maryland.
ELECTROCARDIOGRAMS DURING RESUSCITATION

Results

INITIAL COMPLEX

In general, three types of electrical patterns were noted immediately after the ventricular fibrillation was terminated by external countershock. Type A is characterized by the presence of a P wave preceding the QRS complex and includes cases both with and without ST-T segment changes. Occasionally one could not be certain about the presence of a P wave preceding the first recorded QRS complex, but as the base line recovered and some stability returned, it was apparent five to ten beats later that a P wave was present in the initial complex. The patterns designated as type B are those in which the P wave is absent and the QRS complex is not associated with significant ST segment depression (up to 4 mm). Type C patterns are those also without P waves, but with ST segment depression over 4 mm. The frequency of the three types was surprisingly similar, there being 12, 16, and 11 instances of each, respectively (table 1). In addition, there were four animals in which the initial complex could not be placed in one of the three groups listed. Three revealed persistent asystole, and one demonstrated ventricular tachycardia which quickly reverted to ventricular fibrillation.

Having recognized these three variations in the pattern of the initial complex, we then examined the subsequent course to determine whether differences would be observed among the three groups. The criteria studied were the electrocardiograms taken at one hour, the 48-hour survival, and the overall grading as previously described. This last method of appraisal takes into account the rate of recovery of the central nervous system, and the hemodynamic, as well as the electrical, properties of the heart. It is more descriptive and somewhat more quantitative than mere survival or death. By the assignment of grades (zero to three) for each system, it was possible to obtain numerical values which qualified the recovery as excellent, good, fair or poor.

The data recorded in table 1 emphasize the favorable course observed in animals with the type A initial complex. At 1 hour, 67% of the dogs had normal tracings, all were alive at 48 hours, and the recovery was classified as good to excellent in 92%. The prognosis was less favorable, but nevertheless good, in those with type B complexes since 31% had a normal electrocardiogram by 1 hour, 75% had good to excellent recoveries, and 81% were alive at 48 hours. When the initial complex fell into the type C group only 17% had a good return of electrical activity by 1 hour, and only 2 of every 3 animals survived. The appearance of asystole or ventricular tachycardia was associated with a fatal outcome in the 4 dogs which demonstrated these responses after countershock.

EARLY ARRHYTHMIAS

The first hour after defibrillation was frequently beset with bizarre electrical complexes and multiple arrhythmias. Thus, in 33 dogs there were 52 instances of abnormal rhythms, while only 8 animals showed a sinus rhythm from onset. The latter, of course, survived with excellent function. Table 2 lists the various types of arrhythmias observed.

<table>
<thead>
<tr>
<th>Initial complex</th>
<th>Totals</th>
<th>Normal ECG 1 hr</th>
<th>Alive 48 hr</th>
<th>Excellent to good</th>
<th>Fair to poor</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no.</td>
<td>%</td>
<td>no. %</td>
<td>no. %</td>
<td>no. %</td>
</tr>
<tr>
<td>Type A</td>
<td>12</td>
<td>28</td>
<td>8 67</td>
<td>12 100</td>
<td>11 92</td>
</tr>
<tr>
<td>Type B</td>
<td>16</td>
<td>37</td>
<td>5 31</td>
<td>13 81</td>
<td>12 75</td>
</tr>
<tr>
<td>Type C</td>
<td>11</td>
<td>23</td>
<td>2 17</td>
<td>7 64</td>
<td>6 55</td>
</tr>
<tr>
<td>Unclassified</td>
<td>4</td>
<td>9</td>
<td>0 0</td>
<td>0 0</td>
<td>0 0</td>
</tr>
<tr>
<td>Totals</td>
<td>43</td>
<td>100</td>
<td>15 35</td>
<td>32 74</td>
<td>29 67</td>
</tr>
</tbody>
</table>

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Besides the normal animals, two other dogs were not included; one with persistent asystole, and one with a brief conversion to ventricular tachycardia before the return to fibrillation. Premature ventricular contractions were noted in 30 of the 33 animals with arrhythmias and in 8 of these ventricular fibrillation recurred. The incidence was much higher (40%) when the premature ventricular contractions were multiple (three or more in series) than when they occurred singly (13%). However, the prognosis was not significantly different, in that survival was obtained in 87% of those with occasional and in 80% of those with multiple premature ventricular contractions.

In addition to the electrical indications of myocardial irritability, other arrhythmias were noted. The appearance of either atrioventricular block (nine dogs) or idioventricular rhythm (five dogs) was an ominous sign, for about 60% of these animals reverted to ventricular fibrillation. That this fibrillation was related to these specific arrhythmias rather than to some other change was suggested by the nearly comparable distribution of occasional or multiple premature ventricular contractions, both in the dogs that had recurrent ventricular fibrillation and in those that died. The most serious type of arrhythmia appeared to be an idioventricular rhythm, the occurrence of which was associated with four mortalities among five animals. One of the three dogs with auricular fibrillation had a favorable course; and while the two others reverted to ventricular fibrillation, both of these dogs also had A-V block. Thus there is little reason to be concerned about the presence of auricular fibrillation alone. Indeed, one surviving dog with auricular fibrillation also showed A-V block, multiple premature ventricular contractions, and recurrent ventricular fibrillation before the first hour had passed. Paroxysmal nodal tachycardia, even though accompanied by occasional premature ventricular contractions in three animals, was not followed by any untoward events.

In the 12 animals with recurrent ventricular fibrillation, external cardiac massage and countershocking were continued, and in five a normal sinus rhythm was obtained and maintained for 36 hours. Three of these five had had more than one episode of recurrent fibrillation, and in one survivor four episodes of recurrent ventricular fibrillation preceded the final conversion to a sinus rhythm. Thus, although the occurrence of multiple premature ventricular contractions, A-V block, and idioventricular rhythm were associated with a high rate of recurrent fibrillation, when the resuscitative measures were continued vigorously success resulted in nearly one-half of the trials.

RELATION OF EARLY ARRHYTHMIAS TO DRUGS USED

The need for certain drugs to assist in the early recovery period raises the question of whether the arrhythmias noted actually reflect the electrical behavior of the heart during restoration from ventricular fibrillation, or instead represent the response of the heart to the infused drugs. Various arrhythmias are

---

**TABLE 2**

<table>
<thead>
<tr>
<th>Arrhythmia</th>
<th>Frequency</th>
<th>Recurrent ventricular fibrillation</th>
<th>Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature ventricular contractions</td>
<td>no. 30</td>
<td>% 91</td>
<td>no. 8</td>
</tr>
<tr>
<td>Occasional</td>
<td>no. 15</td>
<td>% 45</td>
<td>no. 2</td>
</tr>
<tr>
<td>Multiple</td>
<td>no. 15</td>
<td>% 45</td>
<td>no. 6</td>
</tr>
<tr>
<td>Paroxysmal nodal tachycardia</td>
<td>no. 5</td>
<td>% 15</td>
<td>no. 0</td>
</tr>
<tr>
<td>Auricular fibrillation</td>
<td>no. 3</td>
<td>% 9</td>
<td>no. 2</td>
</tr>
<tr>
<td>Atrioventricular block</td>
<td>no. 9</td>
<td>% 27</td>
<td>no. 5</td>
</tr>
<tr>
<td>Idioventricular rhythm</td>
<td>no. 5</td>
<td>% 15</td>
<td>no. 3</td>
</tr>
</tbody>
</table>

* There were 52 arrhythmias recorded in 33 animals.
said to occur with the use of the catecholamines employed in our resuscitation studies. While in man most of the disturbances are reported to be supraventricular in origin, ventricular arrhythmias are seen in the dog. In both species these abnormalities are usually associated with a rapid rise in blood pressure, and are closely related to dosage of catecholamine. However, the ventricular tachycardia induced by norepinephrine does not lead to ventricular fibrillation, and even in the presence of ventricular ectopic tachycardia, epinephrine does not precipitate fibrillation.

Because of these reports, we re-examined our data to ascertain the relationship between occurrence of arrhythmias, and the time and dose of the drugs administered. In most instances intracardiac epinephrine was given during fibrillation and was followed by a period of massage. Thus it was impossible to determine whether or not the arrhythmias noted immediately after defibrillation were due to the recently administered epinephrine only. We do not consider the evidence sufficient to incriminate the drug because in some instances no arrhythmias were seen, while in others the abnormalities present after epinephrine were present also in animals which did not receive the drug. The other catecholamine, norepinephrine, was given ordinarily in doses (1 μg/kg/min) which according to Szakacs and Mehlman and Maling et al. do not produce significant arrhythmias in normal dogs. However, in 18 of the 43 experiments the low or undetectable arterial pressure after defibrillation led to the administration of norepinephrine. This overshoot of norepinephrine often produced blood pressures in the range of 300/150 mm Hg. Nevertheless, on eight occasions there were no arrhythmias associated with the overshoot, and in five cases a preceding arrhythmia seemed to improve (i.e., diminish in frequency) with the sharp rise in arterial pressure. This leaves five instances in which occasional (in three) or multiple (in two) premature ventricular contractions appeared in conjunction with the rapid infusion of norepinephrine. The three dogs which showed occasional premature ventricular contractions and one of those with multiple premature ventricular contractions also had these findings either before or after the effect of norepinephrine on blood pressure was noted. Thus in 18 instances when this drug was given in what might be considered an arrhythmogenic dose, only one occasion did an arrhythmia appear which was unquestionably related to overdosage with the drug. For these reasons, we suggest that the arrhythmias described in the preceding section reflect accurately the electrical behavior of the heart during the early recovery from the 15 or 30-minute period of ventricular fibrillation, rather than a response to the drugs used in the resuscitation experience.

**EARLY ISCHEMIC CHANGES**

Apart from the occurrence of certain arrhythmias during the early recovery period, we noted changes in the electrocardiograms which indicated myocardial ischemia and injury. This was not unexpected since the animals had been exposed to a 30-minute period of ventricular fibrillation, during which coronary blood flow was severely restricted, and during which the heart was repeatedly traumatized by external cardiac massage. Therefore, it seemed worthwhile to examine the complexes in order to quantitate the changes indicative of myocardial ischemia and injury. These findings might serve to identify those resuscitative measures which were both least injurious and most effective in restoring the animals to normal. For this reason we measured the maximum change in direction of the R, T, and ST segment which occurred during the first hour of recovery from ventricular fibrillation, beginning about three minutes after defibrillation by which time most bizarre complexes had ceased. These measurements were compared with the control pattern for each animal. Only changes greater than 4 mm were considered significant and the direction in which these took was classified as in-
creased when positive, or decreased when negative with respect to the control tracing. All wave forms during this early period of recovery were taken from lead II. These changes were compared with both the subsequent electrocardiograms and the overall grading of recovery, to determine whether the maximal degree of ischemic change seen early during recovery correlated with subsequent events (table 3). The return of electrical activity was classified as excellent when the tracing resembled the control by 1 hour, good when the electrocardiogram was normal by 24 to 48 hours, and poor when either the electrocardiogram was still abnormal at 48 hours or when resuscitation was not possible.

Thirty-two of the 43 dogs were resuscitated and alive 48 hours after the experiment; 17 of these (53%) having shown normal electrocardiograms 1 hour after recovery. Nine additional animals or 26 of the survivors (81%) were normal by 24 to 48 hours, while 6 dogs still had abnormal electrocardiograms at 48 hours and 7 were dead. In 4 other animals which died, persistent asystole or chaotic complexes did not permit evaluation of ischemic changes. Table 3 shows that the predominant change in the R wave was that of decreased voltage (14 of 39 dogs), while that of the T wave was increased voltage (22 of 39 dogs). In 10 dogs of each group this change was greater than 10 mm. However, these deviations of the R and T waves from normal, in either direction, did not differentiate the good from the poor results.

Alterations in the ST segment appeared to be most meaningful. When they remained within the designated normal range, the outcome was good in that 13 of 14 electrocardiograms were back to normal by 24 to 48 hours, and all animals survived. However, when the ST segment was depressed 11 of 24 animals responded poorly and 7 died. In 10 dogs the depression of the ST segment was greater than 10 mm, but the condition of these animals was not significantly worse than that of animals with less marked ST depression. Thus any ST segment depression over 4 mm can be considered unfavorable, although not necessarily ominous. Elevation of the ST segment occurred only once in the 43 experiments.

**Nonsurvivals**

Despite our resuscitative measures 11 of the 43 dogs did not recover from the period of electrically induced ventricular fibrillation. In contrast, when the electrocardiogram showed no arrhythmias during the first hour after defibrillation there were eight survivors, four animals having completely normal tracings at that time. It seemed worthwhile to compare the variables employed in resuscitation, as well as the variations in electrical recovery.
In the study of cardiorespiratory features influencing the rate of massage, duration of systole, arterial pressure, ventilatory cycling, and manual or automatic massage. When the techniques employed with the two groups were analyzed, no significant differences could be found except arterial pressures obtained during the fibrillation period. Among the group with poor results 4 of the 11 dogs had pressures lower than 60/25 mm Hg, while none of the animals in good condition had pressures this low. When the mean arterial pressure was compared with the control pressure for each animal, in 6 of the 11 failures the pressures during massage were 25% of the control value or less, and were greater than 25% in all 8 dogs that recovered optimally.

We have classified 8 dogs as resuscitated optimally not only because they survived and had a quick return of their electrocardiogram to normal, but also because arrhythmias were absent during the early recovery period. As might be expected, the 11 failures were characterized by serious and multiple disturbances of rhythm. Excluding the animal which remained in asystole after countershock, 3 of 10 dogs had multiple premature ventricular contractions, 4 had idioventricular rhythms, and 8 had recurrent ventricular fibrillation. Seven of these 10 dogs had multiple disturbances of conduction. In 7 of the 11 failures the electrical activity during the first hour after defibrillation could be examined for indications of ischemic injury. As with the total group, no significant differences were noted in the R and T waves, but ST segment depression greater than 4 mm was found in all 7 of the "poor" group, while only 25% of the animals in good condition showed this change. Thus the animals which were not resuscitated were differentiated from those with good results by lower arterial pressures during the external massage period, and by multiple arrhythmias and marked ST segment depression during the first hour after defibrillation.

**Discussion**

In studying the cardiorespiratory features that influence resuscitation from electrically induced ventricular fibrillation, we noted differences in the extent to which each animal recovered its cardiac and central nervous system functions. The temporal variations and the need for therapeutic support during the early recovery period permitted a system of gradation beyond that of survival or death. Thus resuscitation could be graded from excellent to poor.

When the electrocardiograms are examined in detail, certain alterations of prognostic significance can be recognized. First, if the initial complex recorded after defibrillation contains a P wave it is reasonable to assume that the heart suffered minimal injury during the period of fibrillation and massage, and consequently conduction was restored, after countershock, through the normal atrial pathways. This impression was supported by the finding that all these animals survived and 92% had recoveries classified as good to excellent. It is interesting that 5 of the 12 dogs with a P wave in the initial complex also showed ST segment depression greater than 4 mm. The presence of the P wave apparently overrides the otherwise unfavorable significance of the ST segment depression. However, when the P wave is absent, ST segment depression assumes importance, for this characteristic differentiates the better from the poorer results. A reduction in the ST segment greater than 4 mm was associated with a normal one-hour electrocardiogram in only one of six dogs and with survival of only two of every three animals.

When the coronary circulation produced by closed-chest massage is marginal, the resuscitation of such a heart is beset with difficulties which are indicated by poor arterial pressures and a deranged conduction mechanism. Improvement in the blood pressure was effected by the use of norepinephrine, but drugs were not used to influence conduction.
Increased coronary blood flow has apparently a rapid and marked remedial effect, for many of the abnormal complexes and frequent premature ventricular contractions which are noted in the first minute or two after defibrillation usually disappear by the third or fourth minute. Some animals have sustained cardiac damage and a few minutes of normal coronary circulation are not sufficient to repair the injury. These animals exhibit not only disturbances of conduction but also alterations indicating myocardial ischemia. Most dogs showed some myocardial irritability, manifested as premature ventricular contractions, and when these occurred in runs of three or more the incidence of recurrent ventricular fibrillation was three times that observed with single premature ventricular contractions. However, survival was not significantly greater in animals with single vs. multiple premature ventricular contractions. Among the arrhythmias noted, atrioventricular block and idioventricular rhythm were the most ominous because 8 of 14 dogs with these arrhythmias eventually reverted to ventricular fibrillation. A much greater mortality (80%) was associated with idioventricular rhythms than with any other conduction disturbance. While changes in the R and T waves could not be correlated with prognosis, however, more favorable recoveries were noted in animals without significant changes in the ST segment than in animals having ST segment depression greater than 4 mm.

Summary

Continuous closed-chest massage (mechanized and manual) and ventilation with pure oxygen were performed for 15 and 30-minute periods, to maintain viability in 43 dogs with electrically induced ventricular fibrillation. External countershocks were applied and 32 of the animals lived more than 48 hours. The return of electrical and hemodynamic functions of the heart, as well as the function of the central nervous system, were graded arbitrarily to characterize recovery from poor to excellent. Electrocardiographic changes (lead II) were studied in detail and compared with the gross result of survival or death, and also with more quantitative characterization of recovery.

When the initial electrocardiogram after defibrillation contained a P wave, the prognosis was most favorable. When the P wave was absent the amount of ST segment depression in the initial complex differentiated the better from the poorer results.

During the first hour following defibrillation, a variety of arrhythmias was noted, as well as varying degrees of change in the R, T, and ST portions of the electrocardiogram. The most serious arrhythmias, in decreasing severity, were idioventricular rhythm, atrioventricular block, and multiple premature ventricular contractions. Recovery was poorest when multiple arrhythmias occurred. Changes in the voltage of the R and T waves could not be correlated with prognosis. However, more favorable recoveries were noted in animals without significant changes in the ST segment than in animals having ST segment depression greater than 4 mm.

References

ELECTROCARDIOGRAMS DURING RESUSCITATION


Electrocardiographic Patterns During Resuscitation After Experimentally Induced Ventricular Fibrillation
DAVID I. MILLER and MARVIN M. NACHLAS

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