Spontaneous Course of ST-Segment Elevation in Acute Anterior Myocardial Infarction

Rainer v. Essen, M.D., Wolfgang Merx, M.D., and Sven Effert, M.D.

SUMMARY The spontaneous course of ST-segment elevation (ΣST) in 24 patients with acute anterior myocardial infarction (AMI) was studied by precordial ST-segment mapping, which was recorded at 2-hour intervals during the first 48 hours after admission. Change of ΣST between two registrations was expressed as mV/hr, and was compared with clinical and hemodynamic parameters, course of MB-CK curve, calculated infarct mass and arrhythmias. After an initial rapid increase, there was a decrease of ΣST, which reaches a plateau-like curve approximately 12 hours after the onset of chest pain. A second new increase of ΣST exceeding a value of 0.6 mV/hr correlates well with extension of necrosis, verified by re-elevation of MB-CK. During the first 2 days, extension of necrosis could be detected in 50% of our patients.

As new ischemic episodes and extension of necrosis in AMI occur frequently and are promptly indicated by an increase of ΣST, the physician should, while monitoring therapeutic interventions, concentrate on such a second increase rather than on a decrease of ΣST (which may occur spontaneously), as has been suggested in most previous reports.

MANY REPORTS HAVE EVALUATED therapeutic interventions in patients with acute myocardial infarction by precordial ST-segment mapping, but only a few have been concerned with the spontaneous course of ST-segment elevation (ΣST) during myocardial infarction by daily ST-segment mapping, there are none with multiple measurements within the first hours after onset of chest pain, when therapeutic interventions are expected to have the best effect on minimizing final infarct size. Therefore, it is necessary to know the spontaneous course of ΣST during this period for accurate interpretation of therapeutic intervention.

After improving the technique of precordial mapping so that accurate measurements within a relatively short period were possible without interfering greatly with the normal activity of the coronary care unit (CCU), we followed the spontaneous course of ΣST closely and redefined its diagnostic value.

Materials and Methods

Twenty-four patients (19 male, five female), ages 26–81 years (mean age 59.5 years), were studied. All were admitted to the CCU within 24 hours (mean 4.8 hours) after the onset of acute chest pain. All had an ECG compatible with the diagnosis of acute transmural anterior myocardial infarction. Patients with initial signs of pericarditis (pericardial friction rub) or complete bundle branch block were excluded.

Precordial mapping was performed with a flexible synthetic plate (32 × 24 cm) containing 48 silver-oxydized copper electrodes. Contact diameter of electrodes was 0.7 cm and interelectrode distance was 3.5 cm. By means of a switchbox, six adjacent electrodes could be selected and recorded by a six-channel electrocardiograph (Mingograf Cardiorex, 6T, Siemens, Berlin). By marking the edges of the plate on the patient’s skin, registration of identical chest points was possible. The first position of the electrode plate was chosen so that the highest ST-segment elevation was in the center of the plate. In the first 24 hours after admission, precordial mapping was recorded at 2-hour intervals and in the next 24 hours at 3-hour intervals.

Evaluation was performed semiautomatically using a computer (Mulbi 3/20 Krantz Computer, Aachen, West Germany) by fixing the registered ECG leads to a x-y-sensor-frame and alternately touching the zero line and ST segment with an ultrasound graften (Grafpen-Koordinatenleser, Science Accessories Corp, Southport, Conn). The T-P segment was chosen as zero line and only in the presence of sinus tachycardia with poorly identified T-P segment was the PQ interval used as a baseline. ST-segment elevation was measured 0.06 seconds after the spike of the S wave. We needed 15 minutes (± 3 min) to evaluate the precordial maps with this method.

Nineteen of the patients were monitored during the first 2–3 days by hemodynamic measurements with a Swan-Ganz thermomulation catheter floated into the pulmonary artery. Cardiac output was measured via the catheter by thermomulation and the pulmonary artery end-diastolic pressure (PAEDP) was substituted for left ventricular filling pressure (LVEDP).

Serial determination of CPK and CK-MB in order to estimate infarct size as described by Shell and Sobel, and verified by Bleifeld et al. was performed in all patients. During the first 10 hours blood samples were taken hourly; during the next 6 hours every 2 hours, the next 24 hours every 4 hours, and in the next 24 hours every 6 hours. If severe chest pain recurred, hourly sampling was recommended.

Patients received analgesics, sedatives, lidocaine, digoxin, nitroprusside and heparin as indicated. Electrolytes were checked daily and in all cases potassium...
onset of chest pain

FIGURE 1. Course of ΣST during the first 48 hours after onset of chest pain in patients with uncomplicated myocardial infarction. After an early rapid decrease there is a plateau-like course of ΣST with only small changes (< 0.06 mV/hr). One patient had initial right bundle branch block (RBBB) which disappeared in the eighth hour.

concentration was found between 3.9–4.8 mEq/1; this did not exceed the normal range. Heart rate and arrhythmias were monitored continuously by a monitor system (Siemens, Berlin) which allowed an automatic recording of all arrhythmias. Protocolation of heart rate and arrhythmias was performed routinely at 2-hour intervals. Statistical analysis was performed with the paired *t* test.

Results

In the 12 patients in group A, we found the course of ΣST shown in figure 1. The findings observed within the first few minutes in animal experiments by Maroko were not seen in our patients. A rapid decrease in ΣST occurred within 6–12 hours after onset of chest pain. In 14 patients (five in group A,

![Graph showing the course of ΣST during the first 48 hours after onset of chest pain in patients with uncomplicated myocardial infarction.](image)

Within the first few minutes in animal experiments by Maroko were not seen in our patients. A rapid decrease in ΣST occurred within 6–12 hours after onset of chest pain. In 14 patients (five in group A, 9 in group B), the early maximum of ΣST level off to nearly the same plateau as do the seven patients with lower early maximum of ΣST.

![Graph showing the end of spontaneous decrease of ΣST as the point where there is no further decrease (or less than 0.2 mV/hr), the plateau-like course is reached 9.6 hours after onset of chest pain (± 3.5 hr). The seven patients with high early maximum of ΣST level off to nearly the same plateau as do the seven patients with lower early maximum of ΣST.](image)

![Graph showing new increase of ΣST: In group A (n = 12) ΣST increase was very small (0.34 ± 0.14 mV/hr, mean ± SD), whereas group B patients had a significant new increase of ΣST (1.38 ± 0.55 mV/hr).](image)
nine in group B) we were able to study this decrease in ΣST in detail. This fall of ΣST, with a mean value of 2.5 mV/hr, varied considerably between individuals (SD = 1.5 mV/hr) (fig. 2).

This decrease was then followed by a plateau-like course. In 12 patients (group A) this course showed little change in ΣST; the maximal increase was very small (0.34 ± 0.14 mV/hr, mean ± sd) (fig. 3). In these patients serial determination of CPK and MB-CK revealed a typical single increase and decrease of CPK and MB-CK curves (fig. 4).

In a second group of 12 patients (group B) ΣST course showed a new increase with a maximum of more than 0.6 mV/hr (1.38 ± 0.55 mV/hr) (fig. 5). All had simultaneously moderate-to-severe chest pain and CPK and MB-CK curves showed a new increase approximately 6 hours later, indicating that this new rise of ΣST was due to a further extension of the infarction (fig. 6). The change of ST-segment elevation related to reinfarction could be detected by routine 12-lead ECG in four cases.

There was a significant difference (p < 0.0005) between maximal increase of ΣST in group A and group B (table 1, fig. 3). Thus, patients with and without infarct extension could be separated by the degree of a new ΣST increase, the value of 0.6 mV/hr forming the distinction line. Our further investigations of the small changes in ΣST in group A patients have shown that a new rise of ΣST of 0.25 mV/hr to 0.6 mV/hr occurred on 14 occasions and that in nine of them this new rise of ΣST was accompanied by severe precordial pain, necessitating treatment with narcotics. Only five of the
14 cases of severe angina in these patients were not followed by such an increase in ΣST.

In 17 patients, serial CPK determination allowed calculation of infarct size. Patients of group A (n = 9) had an average infarct mass of 56.4 g equivalent (± 44.8 g), including a 26-year-old sportsman with a calculated infarct mass of 165 g equivalent. By comparison, patients of group B (n = 8) had an average infarct mass of 61.3 g equivalent (± 37.7 g). In these cases the infarct size calculation included reinfarction.

Only in group A could a correlation between calculated infarct mass and ΣST elevation during the plateau-like course be found. Group B showed no correlation between ΣST elevation and calculated infarct mass (fig. 7).

Twenty patients were hemodynamically monitored. There was no striking difference between the hemodynamic parameters of groups A and B. Although five patients in each group had initial clinical and hemodynamical signs of left ventricular failure (pulmonary rales, gallop rhythm, PAEDP > 18 mm Hg), mean PAEDP in group B was higher than in group A, though not significantly. Cardiac index was almost the same in both groups (table 1).

To investigate the influence of heart rate on ST elevation, we compared heart rate immediately before and after maximal increase of ΣST (table 2). In both groups A and B we noted a significant rise in heart rate (group A, p < 0.05; group B, p < 0.01). Although the rise in group B was higher than in group A, the difference was not statistically significant.

In 10 patients an increase of heart rate of more than 15% was observed (fig. 8), without any change of ΣST and without the patients experiencing new attacks of angina pectoris. There was no major difference in ventricular ectopic arrhythmias in group A and B. In both groups, five patients had an average of more than 10 ventricular ectopic beats or longer ventricular runs (more than three successive beats). In two group B patients, frequent ventricular ectopic beats started directly before clinical signs of infarct extension and increase of ΣST. In another two patients, ventricular ectopic arrhythmias occurred directly after reinfarction. One group A patient and three group B patients...
developed right bundle branch block; two in group B simultaneously showed signs of reinfarction. All patients were in a sinus rhythm; only one group B patient had to be paced for 3 hours, because of atrioventricular junctional rhythm, and during this period the patient went into ventricular fibrillation. After successful defibrillation, sinus rhythm was restored and could be followed by precordial mapping.

### Table 2. Comparison of New Increase of ΣST, Calculated Infarct Mass, Hemodynamic Parameters and Arrhythmias Between Groups A and B

<table>
<thead>
<tr>
<th>Name</th>
<th>Sex</th>
<th>Age (years)</th>
<th>ΣST (mV/hr)</th>
<th>Maximal 2. increase of ST (mV/hr)</th>
<th>Calculated infarct mass (g)</th>
<th>Initial hemodynamic parameters</th>
<th>Blood pressure (mm Hg)</th>
<th>Ectopic beats (≥10/hr)</th>
<th>Runs of ectopic beats</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>PAEDP (mm Hg)</td>
<td>CI (l/min/m²)</td>
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<td></td>
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<td>Group A</td>
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<td></td>
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<td>M</td>
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<td>24</td>
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<td>150/90</td>
<td>1500</td>
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<tr>
<td>BL</td>
<td>M</td>
<td>75</td>
<td>0.4</td>
<td>—</td>
<td>—</td>
<td>140/90</td>
<td>—</td>
<td>—</td>
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<tr>
<td>DJ</td>
<td>M</td>
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<td>28</td>
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<td>185/120</td>
<td>900</td>
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<td>M</td>
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<td>44</td>
<td>20</td>
<td>2.4</td>
<td>120/85</td>
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<tr>
<td>GW</td>
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<td>55</td>
<td>0.4</td>
<td>40</td>
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<td>3.8</td>
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<td>36</td>
<td>20</td>
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<td>150/90</td>
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<td>F</td>
<td>57</td>
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<td>10</td>
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<td>F</td>
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<td>0.25</td>
<td>20</td>
<td>10</td>
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<td>38</td>
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<td>4.8</td>
<td>140/90</td>
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**Mean**

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<thead>
<tr>
<th>Age</th>
<th>ΣST</th>
<th>Maximal 2. increase of ST</th>
<th>Calculated infarct mass</th>
<th>PAEDP</th>
<th>CI</th>
<th>Blood pressure</th>
<th>Ectopic beats</th>
<th>Runs of ectopic beats</th>
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<td>0.34</td>
<td>56.4</td>
<td>16.3</td>
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<td>137/86</td>
<td>1500</td>
<td>120/75</td>
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**SD**

| 13.2 | 0.14 | 44.8                      | 5.42                    | 0.8   | 11.6          | 0.55          | 37.7                 | 6.8                 | 1.1                 |

**Group B**

<table>
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<tr>
<th>Name</th>
<th>Sex</th>
<th>Age (years)</th>
<th>ΣST (mV/hr)</th>
<th>Maximal 2. increase of ST (mV/hr)</th>
<th>Calculated infarct mass (g)</th>
<th>Initial hemodynamic parameters</th>
<th>Blood pressure (mm Hg)</th>
<th>Ectopic beats (≥10/hr)</th>
<th>Runs of ectopic beats</th>
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<td>BR</td>
<td>M</td>
<td>51</td>
<td>1.1</td>
<td>70</td>
<td>—</td>
<td>—</td>
<td>120/75</td>
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<td>—</td>
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<tr>
<td>CG</td>
<td>F</td>
<td>64</td>
<td>0.7</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>130/80</td>
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<td>DM</td>
<td>M</td>
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<td>1.6</td>
<td>20</td>
<td>14</td>
<td>2.8</td>
<td>140/110</td>
<td>—</td>
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<td>DA</td>
<td>M</td>
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<td>2.0</td>
<td>114</td>
<td>22</td>
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<td>66</td>
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<td>150/80</td>
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<tr>
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<td>M</td>
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<td>0.9</td>
<td>114</td>
<td>12</td>
<td>3.1</td>
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<td>JW</td>
<td>M</td>
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<td>140/90</td>
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<td>70</td>
<td>+</td>
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<td>M</td>
<td>81</td>
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<td>—</td>
<td>15</td>
<td>2.0</td>
<td>130/80</td>
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<td>22</td>
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<td>1.9</td>
<td>115/80</td>
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<td>PK</td>
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<td>61</td>
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<td>19</td>
<td>5.4</td>
<td>145/100</td>
<td>80</td>
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<td>PJ</td>
<td>F</td>
<td>77</td>
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<td>105</td>
<td>70</td>
<td>—</td>
<td>105/70</td>
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**Mean**

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<th>Runs of ectopic beats</th>
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<td>63.8</td>
<td>1.38</td>
<td>61.3</td>
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<td>133/87</td>
<td>120/75</td>
<td>—</td>
<td>—</td>
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</tbody>
</table>

**SD**

| 11.6 | 0.55 | 37.7                      | 6.8                     | 1.1   | 11.6          | 0.55          | 37.7                 | 6.8                 | 1.1                 |

**Abbreviations**: PAEDP = end-diastolic pulmonary artery pressure; CI = cardiac index.
Discussion

Precordial ST-segment mapping, introduced by Maroko et al.,12, 30-33 is designed to estimate the effect of therapeutic interventions from which a reduction of final infarct size can be expected. There has been much discussion about16, 34, 35 drawing conclusions from ECG changes to morphological alterations of myocardial cell damage. Animal experiments demonstrated good correlation between epicardial and precordial ST-segment mapping and histological myocardial cell alterations after coronary vessel occlusion.30-33, 36, 37

Recent clinical reports1-11 on precordial mapping for evaluation of therapeutic interventions in patients with acute myocardial infarction have suggested a reduction of infarct size from a decrease in $\Sigma ST$. Some investigators have rejected ST-segment mapping as a method for monitoring the course of myocardial infarction.16, 17, 34 With improved recording techniques, we were able to reevaluate the capabilities and limitations of this method. We found that $\Sigma ST$ declines rapidly after an initial maximum which is probably reached within the first hour and levels off around the 12th hour into a more plateau-like course.

This was confirmed by Maroko, who found in daily mappings a decrease in $\Sigma ST$ during the first and second day.12 The maximal rate of this initial decline shows large variations. The speed of $\Sigma ST$ reduction depends on the value of the initial maximum; furthermore, it depends on the time when the registration is made after the initial maximum.

Thus, in some cases, it seems impossible to draw conclusions from the amount of $\Sigma ST$ reduction to a proportional reduction in ischemia or myocardial necrosis. Due to a large spontaneous variation in the rate of $\Sigma ST$ reduction, by comparing greater collectives with different therapeutic regimes a meaningful difference in the mean rate of initial $\Sigma ST$ decline may be found.

In uncomplicated cases, after rapid initial changes, $\Sigma ST$ exhibits during the second plateau-like phase only minor hourly changes not exceeding 0.2 mV/hr measured with our method. During this period a new increase of $\Sigma ST$ exceeding 0.6 mV/hr was regularly accompanied by severe chest pain and was followed by a second increase in CPK and MB-CK concentration 4-6 hours later. We believe this is strong evidence for the assumption that such a second increase of $\Sigma ST$ means extension of necrosis. When a smaller hourly increase of $\Sigma ST$, between 0.25-0.55 mV/hr occurred, it was usually accompanied by new severe chest pain but not by a further increase of CPK or MB-CK, so that such an increase of $\Sigma ST$ can be interpreted as an indication of enhanced ischemia. While other investigators have concentrated more on a decrease of $\Sigma ST$ by interventions without noting the spontaneous course, our results indicate that more attention should be focused on a second increase of $\Sigma ST$ and on the hourly rate of increase.

Our results show that a new steep increase of $\Sigma ST$ after the initial decline is — at least in the absence of pericarditis — equivalent to extension of necrosis. We found such an increase in 50% of all cases with anterior myocardial infarction we investigated. This percentage has been confirmed by others.14, 27, 38, 39 In patients with cardiogenic shock due to acute infarction, it was recently demonstrated that all patients had further extension of necrosis.40 While it is impossible to reduce the size of a necrosis which has already developed, it is theoretically possible and worthwhile to attempt to limit infarct size. Therefore, the limitation of infarct size is a useful end point in evaluating therapeutic regimes. In patients with anterior myocardial infarction, precordial mapping promises to be
a valuable method enabling prompt and sensitive detection of new ischemia and necrosis. In animal experiments it has been shown that accelerated heart rate increases ΣST. Our results, however, show that the influence of heart rate seems to be small. Though patients with reinfarction, and therefore steep increases of ΣST, revealed a greater acceleration of heart rate, other patients on many occasions showed an acceleration in heart rate of more than 15% without any significant change in ΣST. However, in this study heart rates were in the range of 70–120 beats/min; rates exceeding 125 beats/min probably have an influence on ΣST, possibly by enhancing ischemia.

There was no significant difference in hemodynamic parameters between patients with and without major second rise of ΣST. Patients with an elevation of ΣST > 0.6 mV/hr and reinfarction (group B) had a slightly higher initial value for PAEDP, but the initial cardiac index was identical in both groups (2.6 1/min/m²). Another question which needs further evaluation is, to what extent the hemodynamic state in the first hour of myocardial infarction contributes to the development of infarct extension.

The occurrence of clinically manifest pericarditis in one case caused surprisingly little disturbance. ΣST did increase, but the hourly increase was small and well under the steep increases of ΣST seen in situations of new ischemia or necrosis. Whether this is true for all cases of pericarditis should be considered.

References

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_Circulation._ 1979;59:105-112
doi: 10.1161/01.CIR.59.1.105
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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