During coronary bypass surgery, the myocardium is exposed to ischemia and reperfusion periods. Thus, the production of reactive oxygen substances increases and oxidative stress occurs.[1] Despite considerable management to protect the myocardium, the ischemia-reperfusion injury still plays an important role in morbidity and mortality of patients undergoing coronary bypass. Several tests are used for determining oxidant status during surgery. We have previously investigated the relationship between ischemia-reperfusion injury and lipid peroxidation, and the stage of coronary bypass in which tissue injury occurs. We found that the coronary sinus levels of lipid peroxidation intermediary and end products, and cardiac marker enzymes were increased at ischemia. These high levels were observed also at all reperfusion periods. Uric acid levels were increased at early reperfusion. However, the total antioxidant capacity level was decreased at ischemia. Uric acid acts as a potent antioxidant and free radical scavenger.[2] According to these observations, we may suggest that myocardial tissue damage occurs at ischemic period and continues at reperfusion stages during coronary bypass.[3]

A plasma oxidizability test is one of the methods for assessment of oxidant status. Kontush et al.[4] reported that the oxidizability of plasma was correlated with the oxidizability of LDL isolated from the same plasma. The utilization of whole plasma offers several important advantages over the assays which use isolated lipoproteins. Most importantly, plasma contains several hydrophilic contents which protect plasma lipoproteins against in vitro oxidation, and which are removed during the LDL isolation. Another important feature of the plasma oxidation assay is that it characterizes the oxidizability of all plasma lipoproteins, rather than those of an isolated lipoprotein class. Other advantageous features of the plasma oxidation assay include rapidness, simplicity, efficient sample processing, avoidance of artifactual oxidation during lipoprotein isolation, and simple photometric registration of the oxidation course.[5-8]
When we performed a plasma oxidizability test in the same patients, we found an interesting result. There was an increasing lag phase, reflecting the resistance of plasma lipids to oxidative modification and a decreasing mean oxidation rate within the lag phase at ischemia and all reperfusion periods (Table 1). These findings suggest that the defensive mechanism is stronger at ischemia and reperfusion period, rather than at the coronary basal status. However, we have demonstrated that oxidative stress was increased at ischemia and all reperfusion periods. The findings of a plasma oxidizability test opposed all other results. In our view, this situation in the plasma oxidizability test may be an interference, and we suggest that a plasma oxidizability test may not reflect the oxidant status in patients undergoing coronary heart surgery. Further investigation is needed to determine which substance interfered with this test.

**REFERENCES**


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**Table 1. The plasma oxidizability test in patients undergoing coronary heart surgery**

<table>
<thead>
<tr>
<th></th>
<th>Coronary basal status</th>
<th>End of ischemia</th>
<th>Early reperfusion</th>
<th>Full reperfusion</th>
<th>Late reperfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lag phase (min)</td>
<td>266.50±55.02</td>
<td>334.33±43.92‡</td>
<td>321.83±67.10‡</td>
<td>344.50±108.10‡</td>
<td>325.17±45.99‡</td>
</tr>
<tr>
<td>Lag phase rate (nmol MDA/min)</td>
<td>0.36±0.17</td>
<td>0.19±0.18‡</td>
<td>0.21±0.15‡</td>
<td>0.20±0.09‡</td>
<td>0.19±0.14‡</td>
</tr>
<tr>
<td>Propagation rate (nmol MDA/min)</td>
<td>1.58±0.78</td>
<td>1.53±0.93</td>
<td>1.68±0.57</td>
<td>1.92±0.84</td>
<td>2.02±0.78</td>
</tr>
<tr>
<td>Maximal malondialdehyde production (nmol)</td>
<td>1.28±0.27</td>
<td>0.94±0.39‡</td>
<td>1.19±0.27</td>
<td>1.12±0.24</td>
<td>1.20±0.24</td>
</tr>
</tbody>
</table>

*Compared with coronary basal status, †: p<0.05; ‡: p<0.01.