We thank you the referee for the constructive and appropriate comments. We changed the manuscript according to the suggestions given that improved significantly our manuscript.

We agree with the reviewer that the effects of gpIIa/IIIb inhibitors have been studied in depth in humans but the experimental models to study the effects on the microcirculation in vivo are limited.

Compulsory revisions:

1. (p.4, 1st para) we agree with the reviewer and clarified the text.
2. We measured many parameters of microvascular damage as increased platelet and leukocyte adhesion, permeability, diameter changes of microvessels and decrease in RBC velocity. These parameters concur to the no reflow phenomenon that has been reported to play an important role in the recovery of the microcirculation. The change in capillary perfusion is the essential parameter in the mechanism of protection, thus determining oxygen delivery to the tissue and permitting the extraction of by products of cellular metabolism during reperfusion. The beneficial effects of t-PA and gpIIa/IIIb inhibition are determined by the increase in capillary perfusion, effect that is derived from a more marked reduction of leukocyte and platelet adhesion but also from inhibition of platelet and endothelial cell activation. We added two representative photographic sequence of microvascular networks treated with abciximab and without any protection against I/R-induced injury. It is evident the lack of capillary perfusion at 15 and 30 min after postschismic reperfusion in a control hamster while the extent of capillary perfusion in the second sequence shows that there is a large recovery of capillary perfusion even if it is not complete.
3. We report the comparison between t-PA with abciximab or eptafibatide vs. abciximab or eptafibatide alone as suggested by the reviewer. The data were already reported in the figures but now we added these findings in the Result and Discussion section. The results obtained in this model indicate that there was still a significant difference in the extent of various microcirculatory disturbances even after gpIIa/IIIb inhibitors compared with abciximab and eptafibatide in combination with t-PA. We give a crucial role to platelet aggregation inhibition while leukocyte adhesion appears to be secondary to the platelets aggregation in arterioles. However, only inhibition of platelet and endothelial cell activation exert complete protective effects against I/R-induced injury because platelet hypereactivity acts as a trigger for events leading to I/R damage amplying all the other mechanisms of damage. We agree with the reviewer that our model is different from AMI patients but all the parameters that we discuss such as leukocyte platelet adhesion are clearly present after 30 min of ischemia and 30 min of reperfusion as shown by many reports in the literature. In our model of I/R it appears that, although abciximab or eptifibatide protected the capillary perfusion, only abciximab or eptifibatide in combination with t-PA caused a significant increase in diameter of arterioles and recovered complete microvascular perfusion.
at the beginning of reperfusion thus indicating that even relatively mild forms of damage may be accompanied by local perfusion deficiencies of the microcirculation and alterations of the endothelial barrier.

4. We gave our manuscript to a native American coworker in order to improve the quality of the writing style.

Discretionary Revisions:

1. P.2, 1st para: We changed the text as suggested: aEoe. Our aim was to compare the effects of abiciximab (Reo Pro) or eptifibatide (Integrilin) alone or with respect to combination with plasminogen activator (t-PA) in an experimental model of ischemia reperfusion (I/R) in hamster cheek pouch microcirculation visualized by fluorescence microscopy.

2. P. 2 3rd para: We introduce in the text the comparison between the combination therapy with t-PA and abiciximab and eptafibatide alone in the Result section. Now we changed the text: aEoePlatelets are crucial in I/R injury, as shown by the treatment with abiciximab or eptifibatide, that decreased platelet aggregation in microvessels, and also leukocyte adhesion in venules. Arterial vasoconstriction, decreased arterial RBC velocity and alterations in the endothelial barrier with increased permeability delayed the complete recovery of perfusion, while the drugs in combination with t-PA accelerated microvascular perfusion after reperfusion.

3. P. 4-5 para: We changed the presentation of the Result section as suggested by the reviewer.

4. P.7,3rd para: We changed the text as suggested.

5. P.7, 4th para: now reported the right reference. We deleted this sentence in the text.