FIGURE LEGEND

E-Figure 1: ***ATP and Purine Metabolism during Ischemia and Reperfusion.***

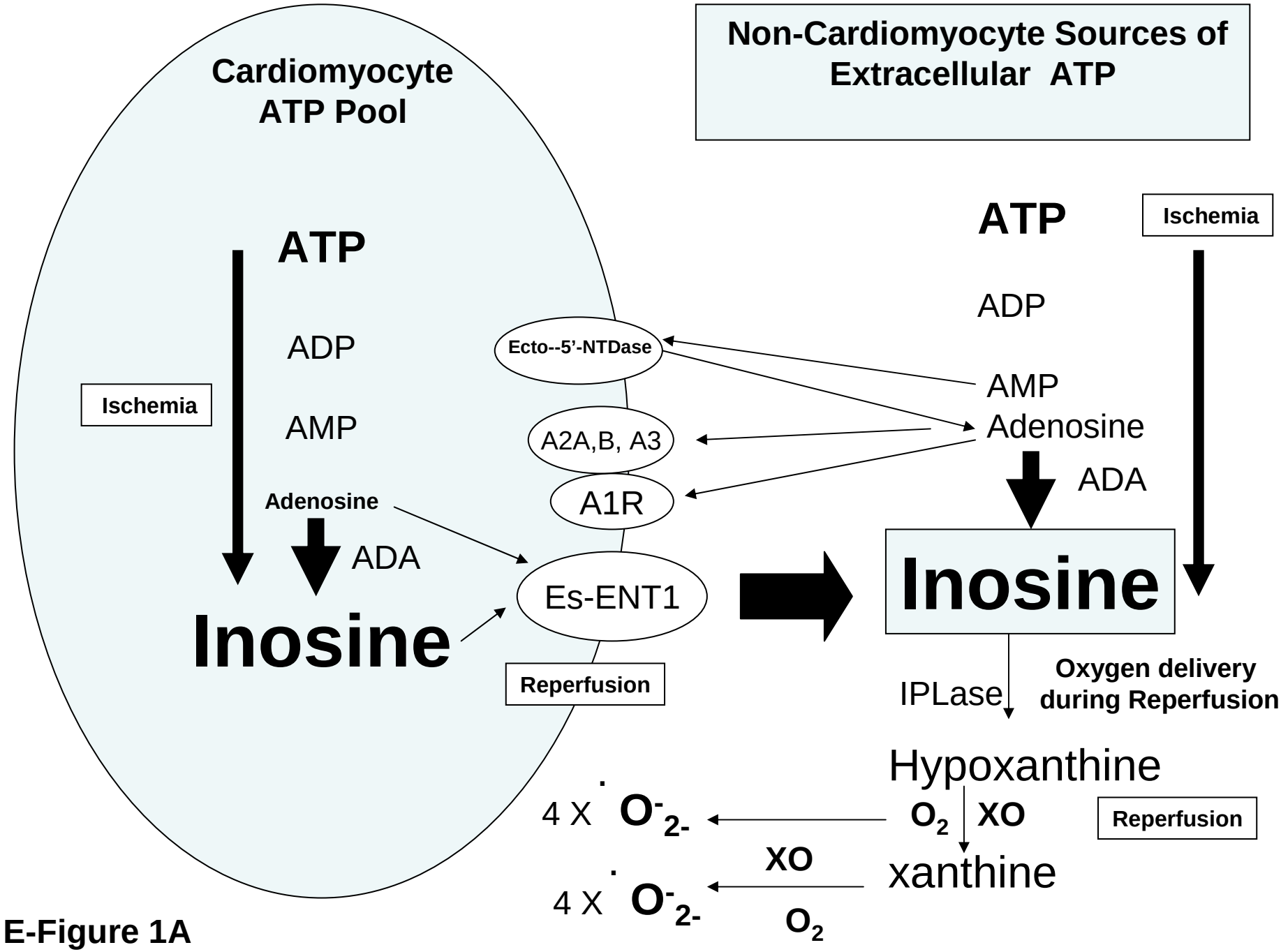
Massive ATP depletion occurs during at the onset of myocardial ischemia. The es-NT1 nucleoside transporter allows the release of both adenosine and inosine upon reperfusion more effectively than during ischemia. Upon reperfusion nucleosides are released and converted to hypoxanthine and xanthine, both are substrates of xanthine oxidase and free radical production (3) and oxidized to free radicals (E-Fig-1A). Non-cardiac ATP co-released from nerve endings with neurotransmitters is also is degraded by ecto-ATPases, nucleotidases and deaminases thus elevating the levels of extracellular adenosine and inosine leading to activation of A1-receptor-mediated signaling mechanisms. E-Fig -1B illustrates that EHNANBMPR inhibits intracellular and extracellular adenosine deaminase activity and blocks es-NT1 transport system thus allowing intracellular accumulation of mainly adenosine during ischemia and reperfusion thus limiting hypoxanthine and xanthine formation and free radical production. E-Fig-1C shows that administration of selective or non-selective adenosine receptor antagonists blocks the action of the extracellular adenosine but not the action of the intracellular adenosine thus interrupting adenosine receptor mediated signaling mechanisms.

Figure 1: ***Effect of 8-SPT and DPCPX on Myocardial ATP Levels***

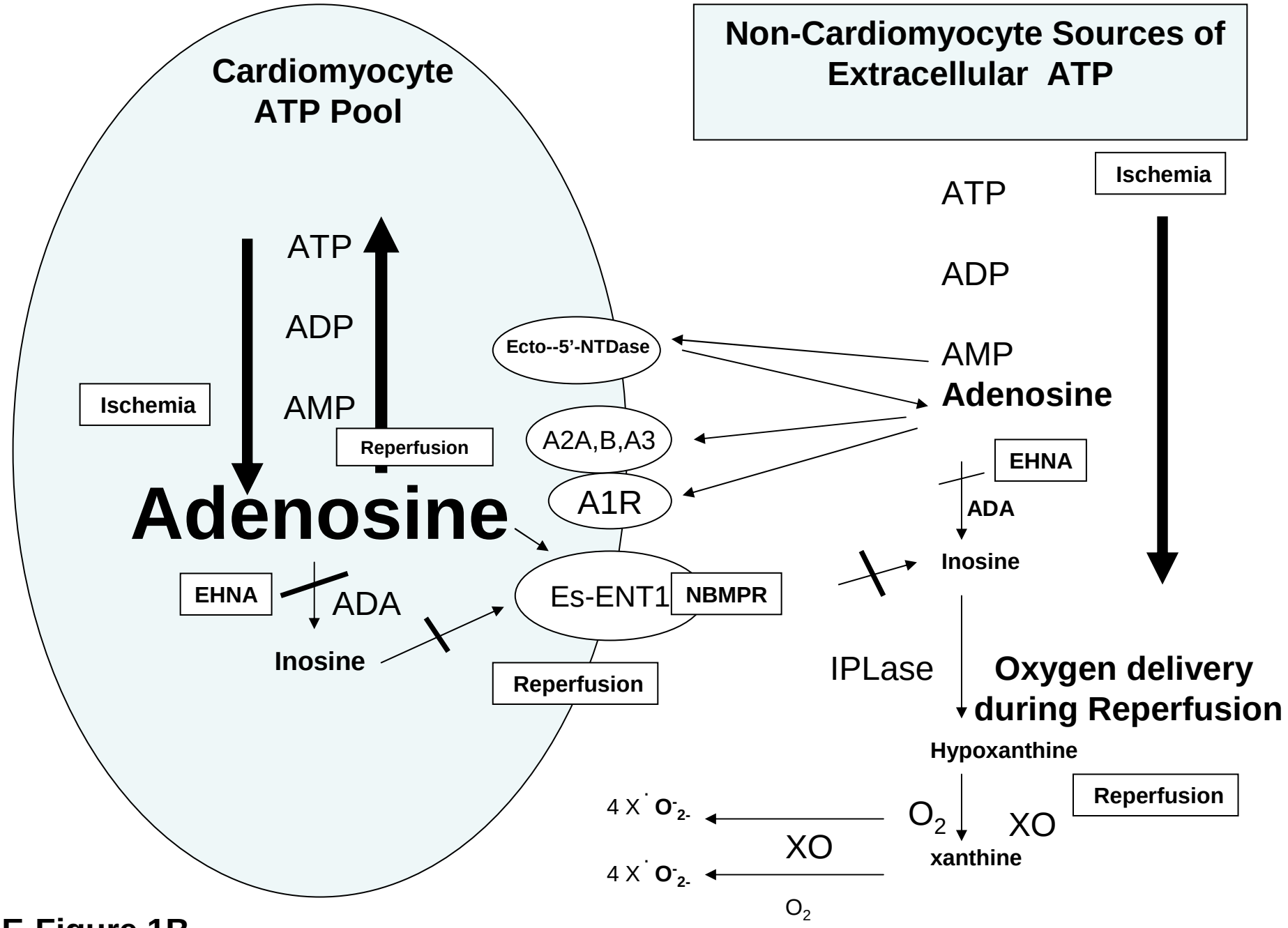
ATP was determined in myocardial biopsies before and after saline (control) or drug administration and at the end of ischemia and during reperfusion. No significant differences between groups with respects to the time of sampling (ANOVA, F=0.04). * Represents significance <0.05 vs. baseline.

Figure 2: **Effect of SPT and DPCPX on Myocardial Adenine Nucleosides Levels**

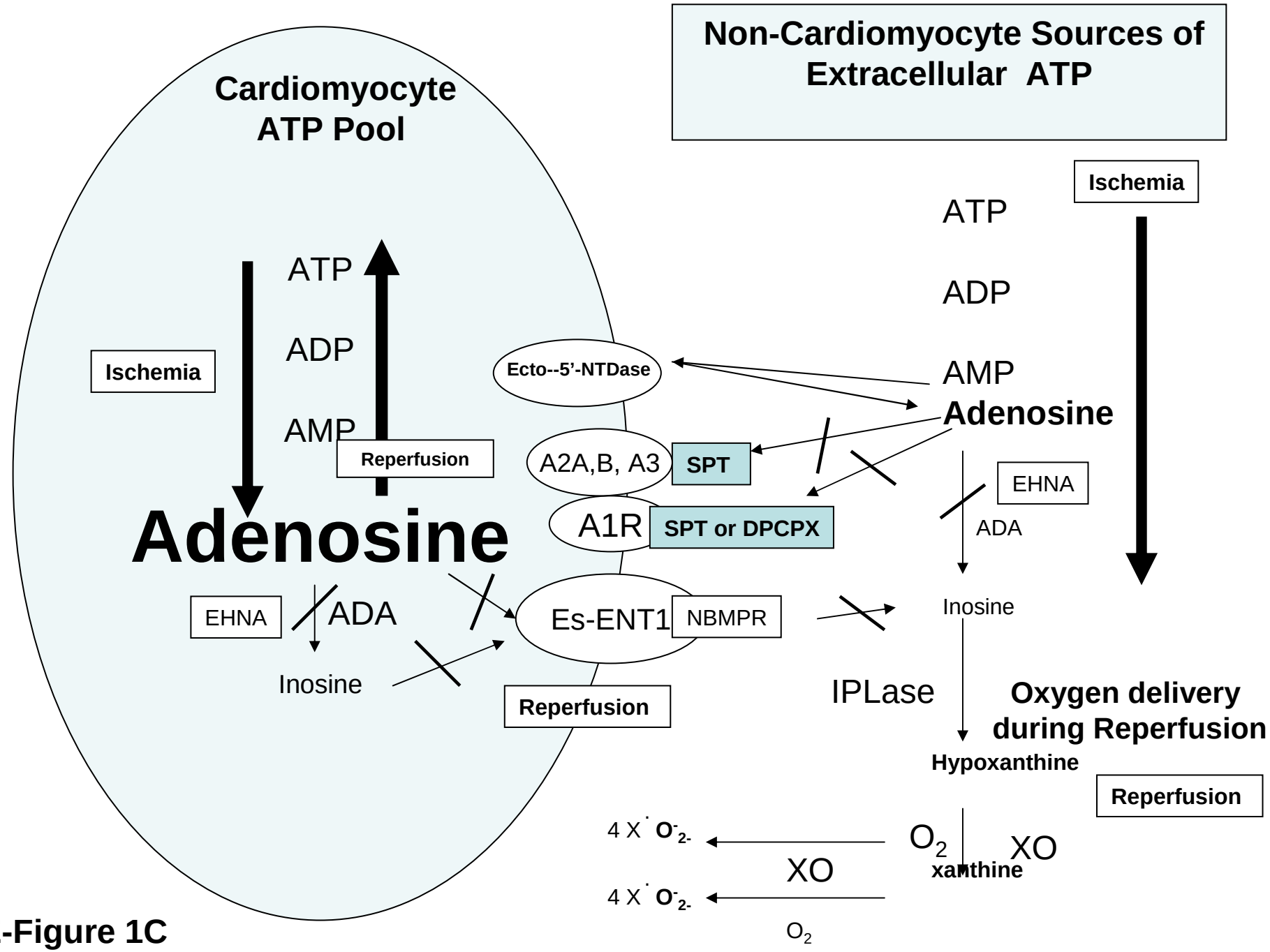
Adenine nucleosides, adenosine (A) and inosine (B) were determined in myocardial biopsies before and after saline (control) or drug administration and at the end of ischemia



E-Figure 1A



E-Figure 1B



E-Figure 1C