**ISCHEMIC PATIENTS UNDERGOING CARDIAC SURGERY HAVE SIGNIFICANT MITOCHONDRIAL COMPLEX I DYSFUNCTION**

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*Background*: Cardiac cells rely heavily on mitochondrial energy production through oxidative phosphorylation. Chronic ischemia may affect mitochondria and myocardial ATP formation altering cardiac function and the bioenergetic state. We present a rapid, reliable and real-time method to evaluate the differences in functional status of respiratory Complexes in mitochondrial isolates extracted from human left atrial appendages (LAA) from patients undergoing cardiac surgery.

*Methods:* Mitochondrial isolates were extracted from LAA in ischemic CABG patients (Group 1) (n=10) and non-ischemic control patients (Group 2) undergoing other cardiac surgery (valve repair/replacement/heart donated for transplant)(n=7). Coupling and electron transport chain assays were performed using Seahorse XFe analyzer. Oxygen consumption rates (OCR) were measured to calculate respiration states.

*Results*: Respiratory control rate (RCR) in Group 1 vs Group 2 was significantly decreased (5.81 ± 0.35 vs 7.51 ± 0.47, respectively)(p<0.01). Absolute respiration significantly declined in Group 1 vs Group 2 (187.8 ± 22.0 pmol O2/minute/µg mitochondrial protein vs 264.2 ± 12.59, respectively)(p<0.05), but there was an insignificant difference for proton leak. Group 1 vs 2 maximal complex I/II respiration ratios were significantly different (58.9 ± 5.47 vs 90.91 ± 8.76 percent, respectively)(p<0.05). There was no significant difference in complex II/IV ratios between groups.

*Conclusion*: Ischemic patients have dysfunctional mitochondria at baseline highlighted by a lowered OCR. This is due to insufficient conversion of ADP into ATP due to Complex I dysfunction or loss. Maintaining or protecting Complex I activity may be a potential therapeutic strategy during cardiac surgery.