Muscle Oxygenation in Children with Congenital Heart Disease

F. Ross, L. Arakaki, W. Ciesielski, G. Latham, D. Joffe, M. Richards, J. Geiduschek, M. Eisses, V. Hsieh, K. Schenkman
Department of Anesthesiology and Pain Medicine, Seattle Children's Hospital
University of Washington

Introduction

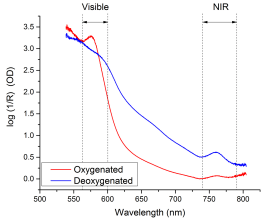
- Background:
  - Children with congenital heart disease (CHD) adapt to chronic cyanosis to maintain tissue oxygenation.[1]
  - Muscle oxygenation can be determined noninvasively using optical spectra of oxy- and deoxy-Hgb and Mgb.[2]
  - During abrupt interruption of O_2 supply, change in Mox during the first minute is directly related to the pre-occlusion steady-state O_2 consumption.[3]
- Research Questions:
  - Do adaptations to CHD result in normal baseline muscle oxygenation?
  - Are there differences in muscle oxygenation and metabolism in cyanotic and acyanotic CHD?

Study Design

- Setup: Optical spectra acquired with a custom fiber optic probe, an LED light source, and an imaging spectrometer
- Data Collection
  - Full spectra from calf or forearm of 49 patients
    - 17 acyanotic CHD (ASD, VSD, Tet, AVSD)
    - 18 cyanotic CHD (TGA, HLHS, DORV, etc)
    - 14 control (+ 20 additional controls for model)
  - CHD group spectra
    - Baseline on 100% O_2
    - 3-min arterial tourniquet occlusion
    - Reperfusion
  - Control group spectra
    - Similar, but with 15 min arterial occlusion for complete desaturation (for model development)
- Spectra converted to Mox using locally weighted regression
- Analysis: CHD groups compared to controls with 2-sample t-tests

Results

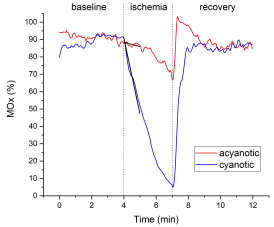
Spectra for Oxy and Deoxy Hgb/Mgb



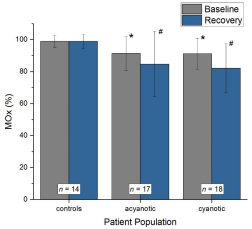
	Controls (n=14)	Acyanotic (n=17)	Cyanotic (n=18)
Baseline MOx (%)	98.9 ± 3.7	91.3 ± 10.6*	91.1 ± 9.7*
Recovery MOx (%)	98.9 ± 4.3	84.6 ± 20.3*	82.1 ± 15.3*
1-min Slope (%/s)	0.17 ± 0.06	0.21 ± 0.24	0.46 ± 0.32*

* Significantly different from controls, two-sample t-test, $p<0.05$

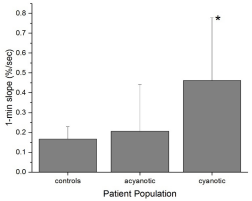
MOx Results for Typical Patients



Baseline/Recovery MOx



Slope of Deoxygenation



Discussion

- Baseline Muscle Oxygenation
  - Patients with CHD (both cyanotic and acyanotic) have lower baseline MOx than healthy patients
  - Baseline MOx is lower in acyanotic patients than healthy controls despite a normal Sp_aO2

Discussion

- Reperfusion
  - Patients with CHD (both cyanotic and acyanotic) had incomplete recovery of MOx after reperfusion compared to controls (despite shorter occlusion)
  - Recovery MOx is lower in acyanotic patients than controls despite normal Sp_aO2 and shorter occlusion
- Oxygen consumption
  - Rate of decline of MOx was greater in cyanotic patients than healthy controls
  - Rate of decline of MOx in acyanotic patients was similar to controls

Both cyanotic and acyanotic patients had low MOx, indicating limited oxygen supply at baseline. In acyanotic patients with normal SpO2, low cardiac output and decreased blood flow to peripheral muscles are plausible explanations for low baseline MOx. Acyanotic patients had 1-min slopes comparable to controls, suggesting normal oxygen consumption. Cyanotic patients had more rapid O_2 decline, suggesting either increased metabolic rate or more likely, limited intracellular O_2 reserves.

Conclusions

This study demonstrates that MOx is abnormal in cyanotic and acyanotic CHD patients. Thus, monitoring MOx perioperatively may provide valuable information in this population.

References

1. Adatia I, Kemp GJ, Taylor DJ, Radda GK, Rajagopalan B, Haworth SG. Abnormalities in skeletal muscle metabolism in cyanotic patients with congenital heart disease: a 31P nuclear magnetic resonance spectroscopy study. Clin Sci (Lond). 1993;85(1):105-109.
2. Arakaki LS, Schenkman KA, Ciesielski WA, Shaver JM. Muscle oxygenation measurement in humans by noninvasive optical spectroscopy and Locally Weighted Regression. Anal Chim Acta. 2013;785:27-33.
3. Gurley K, Shang Y, Yu G. Noninvasive optical quantification of absolute blood flow, blood oxygenation, and oxygen consumption rate in exercising skeletal muscle. J Biomed Opt. 2012;17(7):075010.