# Utilizing Exercise Testing for Critical Decision Making.

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Disclosures: None relevant to this topic.

- Measurements of exercise testing would appear to be ideal endpoints for many clinical conditions and cardiovascular therapeutic interventions.
- Measures of exercise capacity, including aerobic capacity and working capacity, are directly correlated with cardiac output.
- Changes in both maximal and submaximal aerobic performance are associated with clinical cardiovascular health and QOL.
- Exercise performance has been shown to correlate survival and freedom from ACE.

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Modern cardiopulmonary exercise testing (CPET) can be performed at relatively low cost. Measures:

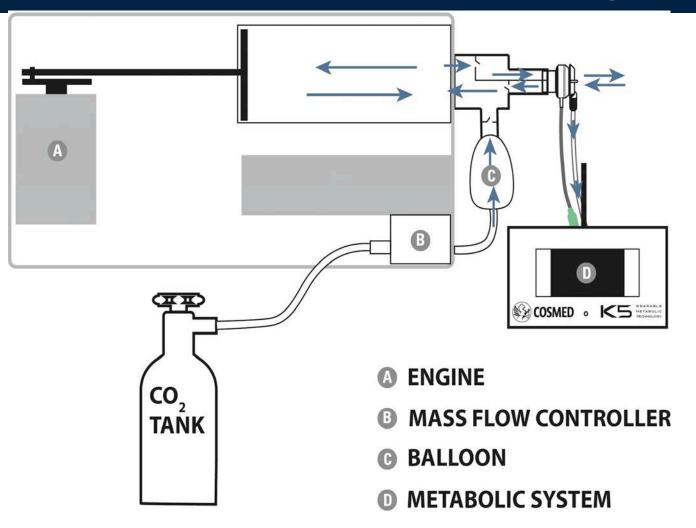
- Gas exchange.
- Cardiopulmonary efficiency.
- Physical working capacity and efficiency.

But, it's a matter of signal and noise.



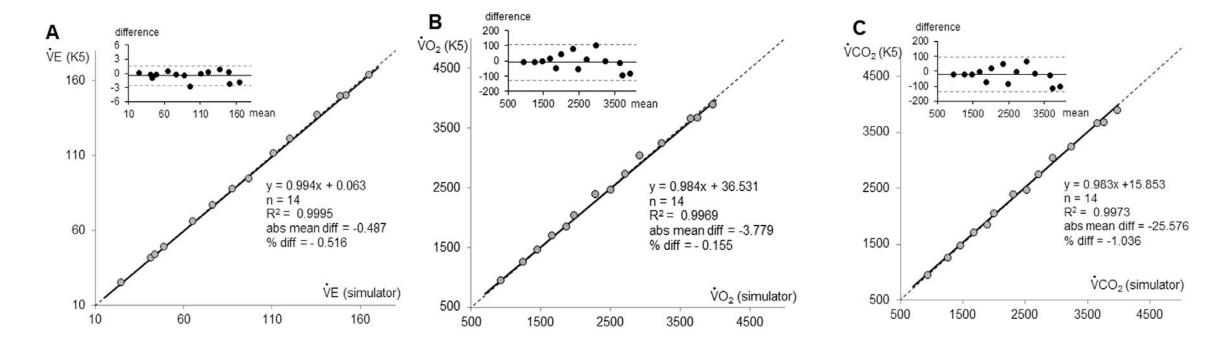
Let's look at how these devices work in theory and practices. The three most important measures are:

- VE
- VO<sub>2</sub>
- VCO<sub>2</sub>



Guidetti et al: <a href="https://doi.org/10.1371/journal.pone.0209925.g001">https://doi.org/10.1371/journal.pone.0209925.g001</a> 2018

Let's look at how these devices work in theory and practices: So far, so good.



Guidetti et al: <a href="https://doi.org/10.1371/journal.pone.0209925.g001">https://doi.org/10.1371/journal.pone.0209925.g001</a> 2018

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What happens when you replace the simulator with a human subject? Under the best of circumstances, there is between a 5 to 10% variance in subjects in test/retest studies.

- Effort.
- Diet.
- Diurnal variation.

Davis et al:Clin Physiol Funct Imaging (2006) 26, pp191–196



- These types of variation in testing are not a problem in healthy, well motivated athletes in which the training effect is likely to far exceed the variance within the measurement devices.
- However, in most cases, this variation will be unacceptably high to allow its use as a standalone test in deciding on a "critical" intervention.
- At best, this type of testing will often be very useful as confirmation in decision making that is being driven by other clinical or laboratory data.

So, if the metabolic measurement have limited utility for standalone "Critical Decision Making", what does?

- Exercise induced ECG changes.
- Exercise induced arrhythmias.
- Exercise induced evidence of myocardial ischemia.
- Rare noncardiac diagnosis presenting as exercise induce symptoms.

Long QT syndrome: In the absence of either resting or exercise induced ventricular ectopy, QT prolongation, especially in mid to late recovery, may dictate initiation of therapy.

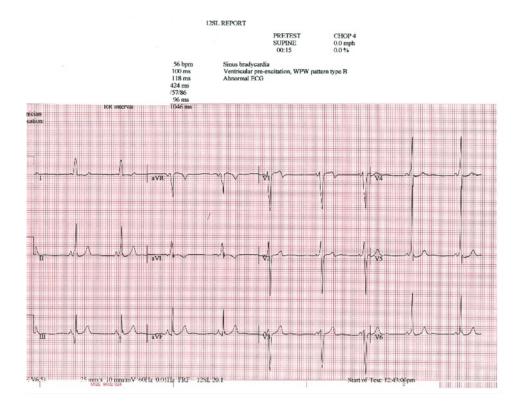
- Resting ECG is normal or equivocal, but history is suggestive of the diagnosis.
- Genetic testing is not available and clinical course would suggest avoiding a prolonged wait for therapeutic decision.
- Genetic testing is available but negative or equivocal.



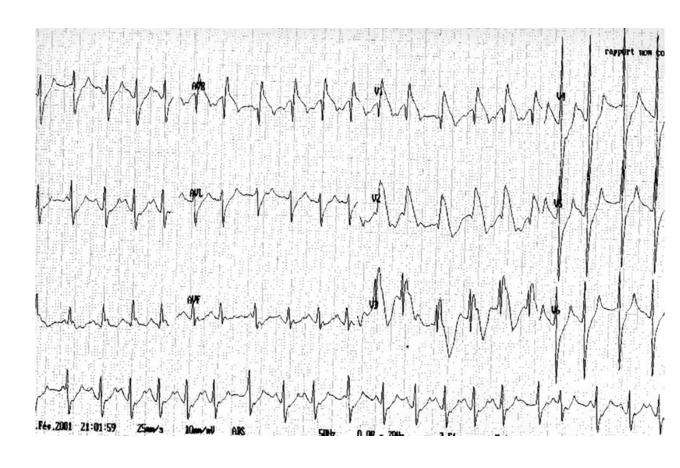
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## Wolff-Parkinson-White Syndrome:

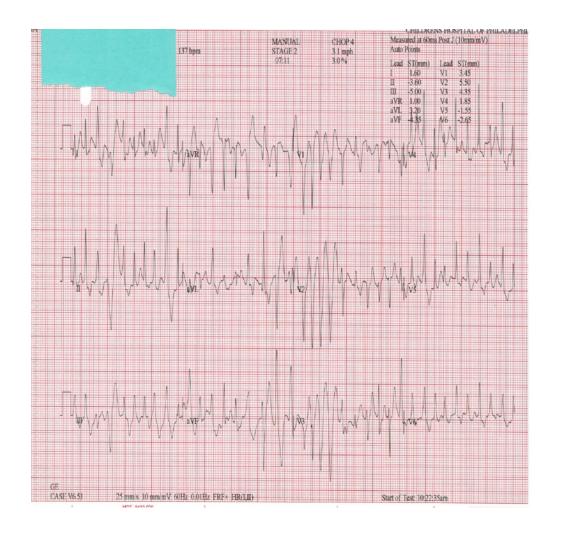
- Resolution of pre-excitation, as risk stratification.
- I'm not sure how useful this is as it seems everyone goes for an ablation anyway.



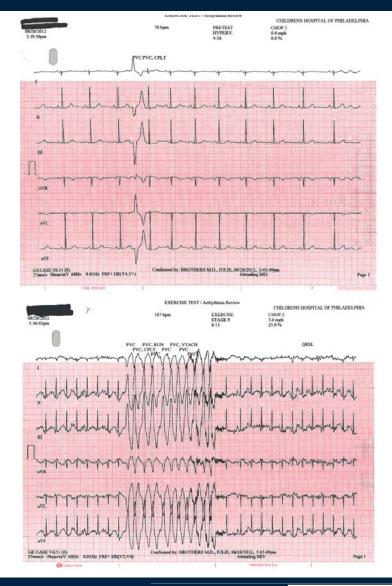
Brugada Syndrome: QRS changes and/or exercise induced ectopy may be useful in diagnosis and risk stratification.



Catecholaminergic ventricular polymorphic tachycardia: Exercise testing is useful for both diagnostic testing as well as for medication titration.



Arrhythmogenic right ventricular cardiomyopathy: exercise testing can be useful for diagnosis in the presence of exercise related symptoms and to monitor effectiveness of therapy.

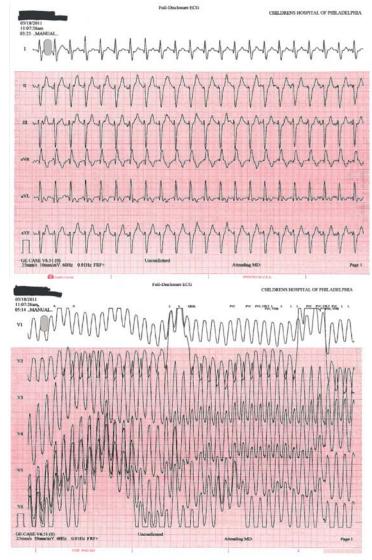




#### Secondary arrythmias:

- Cardiomyopathies, especially HCM.
- Outflow obstructive lesions.
- Operative procedures causing arrhythmogenic scarring.

**DORV** with Melody valve



Identification of exercise induce myocardial ischemia:

- Congenital coronary anomalies.
- Acquired coronary conditions either as a result of a new cardiovascular insult or as a result of an intervention for a non-coronary congenital abnormity.

#### Acquired coronary disease

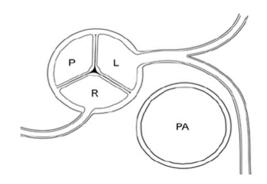
- Kawasaki disease
- Familial dyslipidemias
- Post-op CHD with coronary re-implantation
  Arterial switch for TGA, Ross procedure, aortic root replacement.

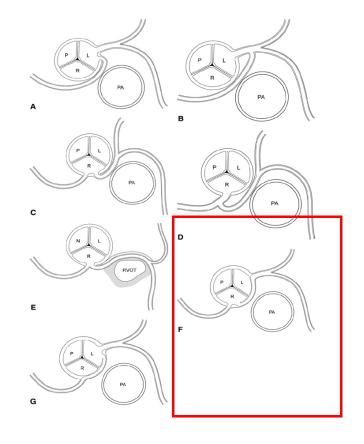
#### Congenital coronary anomalies

- Origin of the LCA from the right sinus of Valsalva
- Origin of the RCA from the left sinus of Valsalva
- Single coronary ostium
- Origin of the LCA from the pulmonary artery
- Ostial stenosis or hypoplasia of either coronary artery

Assessment of stress induced myocardial ischemia is often not clear cut:

#### **Normal Coronaries**

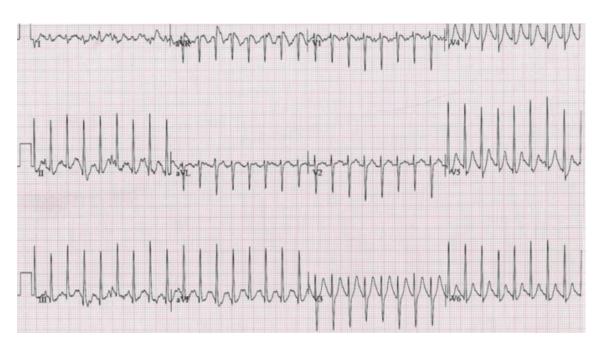




AAOCA left from right with intramural course

The same patient with AAOCA, left from right, tested two weeks apart.



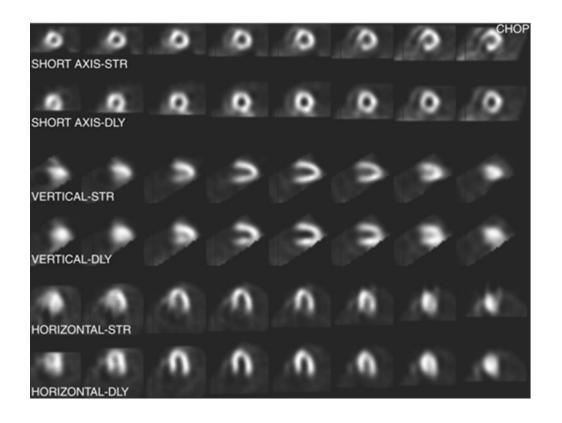


The Journal of Thoracic and Cardiovascular Surgery 2010 140, e27-e29DOI: (10.1016/j.jtcvs.2009.06.029)

Consider adding additional modalities

to assess regional function and ischemia:

- Nuclear perfusion.
- Stress echocardiography.
- Exercise MRI



Finally, the once in a blue moon cases that are usually, but not always, non-cardiac in nature:

- Chiari malformation resulting in vertebral artery compression and syncope.
- Subclavian steal resulting in syncope with upper body exercise.
- Psychogenic symptoms/syncope.

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Thank you.