Measurement of Cardiac Output in the Cath Lab: How Accurate is It?

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Recently, Dr. Herb Aranow, cath lab director at University of Michigan, asked our interventional cardiologist group the following question about cardiac output (CO):

“I have a gentleman who had a bilateral above-the-knee amputation. His height is 104.1 cm and weight is 66 kg. His hemoglobin (hb) is 7.1 mg/dl, due to a lymphoma treated by chemotherapy. His arterial oxygen saturation [sat] was 93% on room air and mixed venous (PA) [pulmonary artery] sat was 57%. Can I estimate his Fick CO/CI [cardiac index] or is it more valid to just report oxygen saturations? In essence, how accurate is our measurement of cardiac output in this setting as well as in the general population of patients?”

To address this, we will quickly review standard methods. Cardiac output (CO) is determined by one of two techniques:

1. Fick technique with measurement of oxygen consumption, or
2. Indicator dilution technique (thermodilution [TD] using a PA catheter).

The TD method is discussed in detail in The Cardiac Catheterization Handbook, 5th ed., and provides information on the theory and practice of obtaining an accurate TD CO.

The Fick principle for cardiac output

The Fick principle states that uptake or release of a substance by any organ is the product of the arteriovenous (A-V) concentration difference of the substance and the blood flow to that organ. The substance in this case is oxygen in blood, but could be other types of indicators as well. As the blood moves slowly, more oxygen is taken out of circulation and the saturations drop, and vice versa.

CO is calculated as oxygen consumption divided by the arteriovenous oxygen concentration difference (in milliliters of oxygen). Remember O₂ saturation percentages are converted to amount (or content) of O₂ using the following formula: content (1.36 x hemoglobin x O₂ saturation x 19).

In the past, oxygen consumption was measured by a hood in which an oxygen sensor cell measured the oxygen content of expired air. The metabolic rate meter reads a cutoff of oxygen consumption in liters per minute. Those devices provided the most accurate measure of oxygen consumption and hence, the most accurate Fick calculations, but are now rarely used. The use of large Douglas bags to collect all expired gas for analysis is of historical interest only.

Cardiac output (CO) using the Fick principle (O₂ consumption) is calculated as follows:

\[ CO = \frac{O_2 \text{ consumption (ml/min)} / A-Mixed Venous O}_2 \text{ difference (ml O}_2 / 100\text{ml blood}) x 10 \]

Problem #1 with the Fick calculation: use of correct oxygen content

The arteriovenous oxygen (A-VO₂) difference is calculated from arterial x mixed venous (PA) O₂ content, where O₂ content = saturation % x 1.36 x hemoglobin. For example, if the arterial saturation is 95%, then the O₂ content = 0.95 x 1.36 x 13.0 g = 16.7 ml PA saturation is 65%, and O₂ consumption is 210 ml/min (70 kg x 3 ml/kg) or measured value, and CO would be determined as follows:

\[
\begin{align*}
\text{CO} & = \frac{210}{0.95 - 0.65} \times 1.36 \times 13.0 \times 10 = 396.56 \text{min}^{-1} \\
\end{align*}
\]

Problem #2 with the Fick calculation: Assumptions about accurate oxygen consumption

To address the question raised by Dr. Aranow, I wanted to share the conversations regarding how to best calculate CO from our experts.

Mort Kern, Long Beach, CA: You can estimate CO from the most commonly used formula, 3 ml O₂/kg, but this formula is not very accurate when compared to any direct measurement. There are several alternate formulas proposed (see Table 1), but also weakly correlated to measured Fick (Narang et al). Andrew Doorey and colleagues from Delaware published in CCI on the better formulas. Since the variability of CO with the standard formula is so large, we report estimated Fick, O₂ sats, and when measured, TD outputs. We may all accept that for general purposes, the clinical difference between an output of 3 and 4 l/min has little real meaning for decision-making. Based on PA sat 57%, continued on page 6

![Figure 1](image_url)
Cardiac Output

Continued from page 4

I’ll bet his ejection fraction (EF) is low and output low.

Andrew Doorey, Newark, DE: Here is the plotted data from our study that Mort referred to above (Figure 1). The assumed Fick gave particularly bad results (as has been reported many times, once we reviewed the literature) and many feel it should be abandoned. In fact, our system uses the LaFarge equation. This equation is based on pediatric measurements, and is specifically recommended not for use in adults. With this system, if you enter the sat before the TD, the assumed Fick will be the default for the report and any valve calculations, unless you do 11 non-intuitive steps to change it. Clicking on the TD button only changes how it is displayed on the screen. Many don’t know this and it is not in the software documentation.

We use the same VO_{2} (i.e., O_{2} consumption) system as Mayo (Figure 2) that we borrow from the cardiopulmonary lab. Way easier to use than the Douglas bag. Some minimal training is required, but we found some pretty big differences between ‘assumed’ Fick, and even TD and the actual (we called it ‘measured’ in our paper) Fick. This could possibly be a big deal when calculating valve areas.

Lloyd W. Klein, Chicago, IL: I am having trouble with the idea that amputation increases CO. It makes total sense to me that amputation would decrease VO_{2} as stated earlier, and also that at rest, O_{2} extraction would be low compared to during exercise. It makes no sense to me that amputation would increase CO; I can see no effect or a small decrease. Therefore, leg/muscle extraction even at rest must be higher than Mort thinks or else he would be correct, which seems paradoxical to me. [Clin. editor’s note: That I’m correct or the CO is high?]

I searched Google. As you can imagine, most studies are mainly interested in critically ill patients who are critically ill, available online at http://www.ncbi.nlm.nih.gov/pubmed/20418366. Femoral vein sats are slightly lower than mixed venous sats, and don't change proportionately with mixed venous O_{2} in critically ill patients.

Now, the next assumption would be that having no legs has no impact on oxygen extraction or flow through the viscera. If we assume that, I think we can explain then how amputation might affect the Fick equation. But honestly, I really don’t know any of this for sure.

I am at least as much concerned about how the hematologic disease affects oxygen-carrying capacity in this patient as the amputation. It is possible that there is less affinity for O_{2} and that the relationship in the assumed Fick is entirely fallacious for this case.

Barry Borlaug, Minneapolis, MN: Figure 3 shows the distribution of cardiac output (matched to local VO_{2} at rest (bottom) and during exercise (top). Skeletal muscle normally receives 15-20% of CO, but doesn’t extract much, as Mort points out, because there is little demand in the absence of muscle contractions. With exercise, skeletal muscle receives an absolute and proportional increase in flow, which is also met with enhanced extraction. There is vasoconstriction to regions where increased flow is not needed (kidneys, splanchnic vasculature). In fact, if this did not occur, we’d all become hypertensive and pass out with exercise (this is what happens in rare splanchic ganglionopathies where this regional vasoconstriction to allow for perfusion redistribution is lost).

At Mayo, we measure VO_{2} directly at the time of PA and arterial blood sampling using a metabolic cart made by MGC Diagnostics (Figure 2). This is exactly the same device that is used in cardiopulmonary exercise testing in our lab. A bit of up-front expense and training of staff is required, but it is pretty straightforward and saves “doctor time” with not having to wait around and shoot 5 thermodilution outputs. My opinion, given the error with assumed VO_{2}, is that if you really need to know accurate CO and you cannot measure VO_{2} directly, you should do TDICO.

Mort Kern, Long Beach, CA: The lowest venous sat is from the venous drainage of the coronary sinus, then the head. Higher sats come from renal veins (not much extraction). While the lower extremities make up about 35-40% body mass, they are not extracting O_{2} at rest and thus the venous sat at rest is about the same at IVC [inferior vena cava] near the heart. When active, the muscle extraction of O_{2} and sats drop. My guess would be that amputations would reduce O_{2} consumption (less mass to consume O_{2}) and thus the calculation for CO would be higher than before amputation.

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Kirk N. Garratt, New York City, NY: OK, I buy that VO_{2} isn’t affected by the anemia, but am having a hard time buying that the amputations don’t affect it. Lower extremity muscle mass has high metabolic demand, maybe the highest of any muscle groups, so losing both legs has got to lower VO_{2}. Wiki-Answer (the ultimate research resource) says your legs account for about 40% of your mass, but it is a mass with high O_{2} extraction. I think the standard calculations for output should be accurate as long as you’ve got an accurate (corrected) BSA [body surface area] and a measured VO_{2}. Estimated VO_{2} values will, I think, have even higher error than usual because the lost mass was enriched in high-O_{2} consuming tissue.

Lloyd W. Klein, Chicago, IL: Those old papers are great. I am just not sure still whether bilateral AK [above knee] amputation is relevant. As you know, where you do the IVC sat matters in terms of the renal vein stream, as there are differential rates of O_{2} extraction in visceral versus muscular beds. I am speculating here, that if there are no legs, then all the IVC return is visceral and thus a truly “indeed” IVC sat could be lower without legs. That might explain, to some extent, the low PA sat in your case. Now, does having no legs diminish O_{2} consumption? Your papers suggest not. Is CO, or CI, diminished? Again, my guess is that although still in the normal range, it must be 10-20% lower, because the lower extremities get that

Table 1. Alternative formulas for calculating cardiac output.

<table>
<thead>
<tr>
<th>Formula</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Dehmer formula</td>
<td>VO_{2} (ml/min) = 125 × BSA</td>
</tr>
<tr>
<td>2. LaFarge formula</td>
<td>VO_{2} (ml/min) = 138.1 – (11.49 × log age) + (0.378 × Heart Rate) × BSA (Men); VO_{2} (ml/min) = 138.1 – (17.04 × log age) + (0.378 × Heart Rate) × BSA (Women).</td>
</tr>
<tr>
<td>3. Bergstra formula</td>
<td>VO_{2} (ml/min) = 157.3 × BSA + 10 – (10.5 × log age) + 4.8 (Men); VO_{2} (ml/min) = 157.3 × BSA – (10.5 × log age) + 4.8 (Women).</td>
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</tbody>
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Note: O_{2} consumption is referred to as “VO_{2}” in this table.
Cardiac Output

Continued from page 6

much CO normally. Maybe there is some compensatory mechanism at work.

Herb Aranow, Ann Arbor, MI: A study by Gailey et al that compared resting oxygen consumption in 39 patients s/p status post traumatic BKA [below the knee amputation] (not exactly the same as my patient who was s/p bilateral AKA for severe PAD [peripheral arterial disease], but the best data I could find). vs. 21 normal controls suggests there was no difference in resting VO₂ between these 2 groups. Another paper by Colangelo et al provides resultant reductions in BSA following amputation of various limb segments/digits, stratified by gender. Perhaps one could use this table to convert PI CO to CI of needed? Finally, Narang et al compared measured VO₂ using the Douglas bag technique to derived VO₂ using 3 different formulae (see Table 1). As Bourlaug and Narang et al note, an error in derived vs measured VO₂ may be >25%. So, that begs the question, what is the current best practice for determining PI CO/CI in the cath lab? I can tell you that we are NOT using a Douglas bag in my lab.

Barry Borlaug, Minneapolis, MN: [The anemia is not related to O₂ consumption.] No relation between total body VO₂ and Hgb, unless there is secondary increased myocardial O₂ consumption to maintain higher output state with anemia. Error of assumed VO₂ may be +/-25% in this circumstance. The A-VO₂ difference (Fick denominator) is:

\[(93-57)*1.34*10/7.1 = 34.3\]  

This is not that different from what you would get with normal PA sat (75%) and a normal Hgb (14):

\[(93-75)*1.34*10/14 = 33.8\]

So if VO₂ is normal, then this anemic CO is pretty normal output, but that is still a big “if” without direct VO₂ measurement.

Mort Kern, Long Beach, CA: How do you estimate O₂ consumption based on Hgb? High Hgb, low consumption? PA sat of 57% despite anemia suggests low CO. I should think.

Larry Dean, Seattle, WA: Not so sure. With the anemia I suspect the assumed PI will not be low. Quick calculation is 4.7 l/min and CI 3.4 l/min/m².

Sam Butman, Valle Verde, AZ: Even more so (low EF or at least a low CO) given his quite significant anemia, which should be giving him a much higher PA sat.

Navin Kapur, Boston, MA: To go back to the original question, a few thoughts:

1. For inpatients with uncompensated heart failure, we often track mixed venous sats and arterial sats in our heart failure patients without necessarily following PI or TD outputs. This allows us to titrate therapy in response to acute hemodynamic changes with our original dataset as an internal control.

2. The B-cell lymphoma also complicates issues here both on the arterial sat and the venous sat. Not sure I would rely much on any of the saturation measures other than to rule out shunt, evaluate his pulmonary function, and to track for acute changes depending on why the patient was cathed in the first place.

3. If we really want a sense of this patient’s cardiac function and reserve function, then cardio-pulmonary exercise testing with measured VO₂ is likely the best way to go. All bets are off when trying to correlate invasive hemodynamics and LV [left ventricular] ejection fraction (which should be primarily used to stratify him as HFpEF [heart failure with preserved EF] / HFrEF [HF with reduced EF] and to identify his candidacy for device therapy [implantable cardioverter debrillator (ICD), etc.].

Mike Ragosta, Charlottesville, VA: The hemoglobin doesn’t influence the oxygen consumption; that’s the demand side while the anemia is the supply side. The low PA saturation in the presence of anemia does not necessarily imply low cardiac output. If you recall Dr. Grossman’s analogy, if you think of the red blood cells as the “trucks” carrying the oxygen, for any given oxygen consumption, if you have anemia, you have less “trucks” delivering the oxygen. Thus, to meet the oxygen demand, you will need to extract more oxygen from the fewer trucks and thus you will have a lower mixed venous saturation in the setting of anemia as compared to the same cardiac output without anemia. Seems like you have to actually measure oxygen consumption in this case. Otherwise, you are just guessing.

From Narang et al’s Circulation publication: “…measured VO₂ differed significantly from values derived from formulae of Dehmer, LaFarge, and Bergsma [Table 1] with median (interquartile range) absolute differences of 28.4 (13.1, 50.2) ml/min, 37.7 (19.4, 63.3) ml/min, and 31.7 (14.4, 54.5) ml/min, for the respectively (P < 0.0001 for each). The measured and estimated values differed by >25% in 17% to 25% of patients depending on the formula used. Median absolute differences were greater in severely obese patients (body mass index > 40 kg/m²), but were not affected by sex or age.”

The bottom line

I agree with Drs. Borlaug, Ragosta, and Narang and colleagues, that “…estimates of resting VO₂ derived from conventional formula are inaccurate, especially in severely obese individuals. When accurate hemodynamic assessment is important for clinical decision-making, VO₂ should be directly measured.” It looks like we’re all going to need an O₂ consumption hood.

Acknowledgment

I want to thank my colleagues from across the country, true experts and teachers, who have so thoughtfully contributed to this informative and entertaining educational discussion. My great appreciation to you all.

References
