Reply to the Editor:

We appreciate the comments of Drs Bonde and Graham regarding the surgical management of massive pulmonary embolism. To improve visualization of the pulmonary artery tree, they used cardiopulmonary bypass with aortic clamping and cardioplegia. They reported their experience in 3 patients.

We believe aortic clamping and cardioplegia are not necessary, because reductions in flows have been brief in duration and this has provided adequate visualization in the majority of cases. One problem with their approach will be right ventricular dysfunction. Most patients have some degree of postoperative right ventricular dysfunction, and aortic clamping will certainly make this worse. In our series, several patients demonstrated postoperative right ventricular dysfunction necessitating inotropic support *despite* our practice of avoiding aortic clamping.

In summary, we believe the risks of right ventricular dysfunction imposed by aortic clamping and cardioplegia are greater than the benefits of improved visualization in the occasional case.

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Assumed oxygen consumption in the determination of cardiac output:

Assume carefully . . .

To the Editor:

With great interest we read the article of Fakler and coworkers¹ in which they warn of large errors in determination of cardiac output when using the Fick principle with assumed oxygen consumption (VO_2).¹ We share the concerns of the authors, especially in specific patients with potentially deviating VO_2 compared with a control group, such as children with congenital heart disease.^{2,3} However, methodological errors in the study of Fakler and associates may surpass the error introduced by the use of assumed VO_2 in these patients.

Although we hope that our major concern is based on typographical errors, we fear a major flaw in the analysis in this study. According to the described equation in the Methods section, the assumed VO_2 values derived from the LaFarge

and Miettinen equations are being divided by body surface area (BSA) to reach indexed values. However, LaFarge and Miettinen⁴ derived their equation for indexed VO_2 . In other words, the presented equations are incorrect and will lead to highly erroneous values, resulting in false high values in smaller patients ($\text{BSA} < 1 \text{ m}^2$) and false low values in larger patients ($\text{BSA} > 1 \text{ m}^2$). In their original study, LaFarge and Miettinen⁴ included over 800 patients and found indexed VO_2 values between 112 and $162 \text{ mL} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$. From Figure 4 in the current article, it can be estimated that in this study more than 70% of the $\text{VO}_{2\text{LaFarge/Miettinen}}$ values are outside this range, whereas the authors claim that the studied patients were in the same age range as the original LaFarge and Miettinen population. This strongly suggests that the authors indeed applied a wrong equation to assume indexed VO_2 . In this population with a mean age of 11.3 years, this could be one explanation for the large overestimation of assumed VO_2 .

By analyzing indexed values of VO_2 instead of the measured quantity, the authors make it impossible to analyze whether the occurring errors correlate with the absolute magnitude of VO_2 . In general, this should be done when comparing measurement methods for accuracy.³

Second, it can be questioned whether absolute values of VO_2 determined with the Deltatrac II system (Datex, Engström, Helsinki, Finland) are acceptable as a gold standard. Although this system has been validated in vivo, this was mainly for its use in metabolic studies, requiring accurate and stable respiratory quotients. It has not been well validated for absolute values of VO_2 in mechanically ventilated children, in whom the prevention of air leakage is extremely cumbersome but imperative for accurate measurement of absolute values.

Finally, in contrast to the statement of the authors, in patients with congenital heart defects, adequate values for VO_2 are not necessary for the determination of shunt ratio.

In conclusion, we absolutely agree with the authors that routine use of assumed VO_2 may result in errors in the determination of Fick cardiac output. Unfortunately, we question whether the presented data can be used to support this common opinion.

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Reply to the Editor:

With great interest we read the comments of Berger and Bergstra, and we appreciate their remarks concerning our article.¹ We have to confirm with regret that there is a typographical error in the published version of our article. The correct version of the LaFarge/ Miettinen formula² is as follows:

For females: $\text{VO}_2/\text{BSA} = (138.1 - 17.04 \times \ln(\text{age}) + 0.378 \times \text{HR}) (\text{mL}/\text{min})/\text{m}^2$

For males: $\text{VO}_2/\text{BSA} = (138.1 - 11.49 \times \ln(\text{age}) + 0.378 \times \text{HR}) (\text{mL}/\text{min})/\text{m}^2$

However, we used this correct version for the analysis of our data, so the data and figures we presented are correct.

The difference between the populations and the regimen of general anesthesia and relaxation might explain the different findings of assumed oxygen consumption (VO_2) values.

Secondly, Berger and Bergstra questioned whether the determination of VO_2 with the Deltatrac II system (Datex, Engström, Helsinki, Finland) is acceptable as a reference method.

Behrends and colleagues³ showed an acceptably accurate determination of VO_2 using the Deltatrac II system compared with mass spectrometry and wet gas spirometry in an in vitro model for ventilated neonates: mean bias -3.8% (SD 5.7%).

Weyland and colleagues⁴ described a laboratory validation in a pediatric setting comparing the Deltatrak system with mass spectrometry and wet gas spirometry. They found a mean bias of -3.2% (SD 11.5%), including the following conditions: F_{IO_2} (inspired oxygen fraction) 0.21 to 0.8, F_{EO_2} (expired oxygen fraction) 0.01 to 0.05, V_E (expired volume) 300 to 6000 mL/min, V_T (tidal volume) 8 to 300 mL, Paw (airway opening pressure) 10 to 60 mbar, and relative humidity 10% to 60%.⁴ These wide ranges of respiratory variables might explain the wider ranges of the standard deviation.

Bauer and colleagues⁵ performed an in vitro validation and clinical testing for ventilated preterm infants. In vitro validation was done by methanol burning during intermittent positive-pressure ventilation (IPPV) with two commonly used ventilators: Sechrist IV 100B (Sechrist Industries, Anaheim, Calif) and the Infant star software version 83 (Nellcor Puritan Bennett Inc, Carlsbad, Calif). They found a mean difference between VO_2 measurements using the Deltatrak II system and VO_2 determined by the methanol burning method from -2% to 2% (max SD $\pm 5\%$) depending on the ventilator and peak inspiratory pressure they used.

For clinical testing, they developed a breath collector to avoid gas leakage around uncuffed endotracheal tubes or during nasal continuous positive airway pressure (CPAP). This test arrangement simultaneously sampled expired air expelled at the ventilator and escaping via the tube leak from the infant's mouth and nose and allowed VO_2 measurements that are not affected by tube leaks.

They studied 15 preterm infants during IPPV and 10 preterm infants during nasal CPAP and determined a mean difference of VO_2 of 6.5 (SD 1.5) mL $\cdot$ kg⁻¹ $\cdot$ min⁻¹ during IPPV and 6.6 (SD 2.2) mL $\cdot$ kg⁻¹ $\cdot$ min⁻¹ during nasal CPAP.

In conclusion, these studies prove that the Deltatrak II system is a feasible and reliable device to measure VO_2 in ventilated infants if air leaks can be excluded.

We determined air leak by measuring inspiratory and expiratory tidal volume using the features of the ventilator (Siemens Servo Ventilator 900 D; Siemens, Erlangen, Germany). As described in the Methods section, we had to exclude 12 patients because of air leakage of more than 5%.

Finally, Berger and Bergstra correctly pointed out that a shunt ratio can be calculated using oxygen saturation measurements. A determination of pulmonary or systemic blood flow is necessary to calculate absolute shunt volumes.

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Triiodothyronine in neonatal heart surgery: Looking for an answer To the Editor:

We congratulate Mackie and colleagues¹ on their randomized study on triiodothyronine (T3) treatment in neonatal heart surgery. In their study group the authors correctly enrolled a homogenous group of infants at highest risk of postoperative low cardiac output syndrome and marked thyroid hormone suppression.² As already shown by several other authors, the thyroid hormone levels are suppressed after cardiopulmonary bypass (CPB), decreasing to a nadir at around 48 hours after CPB and recovering over approximately 1 week. Logically, treatment with T3 after CPB raises the T3 plasma concentrations to normal levels, and no negative effects have been demonstrated so far. Previous studies, as well as

the study presented, had relatively small subject numbers, thereby limiting the ability to determine a clinical effect. This led to the initiation of the Triiodothyronine for Infants and Children Undergoing Cardiopulmonary Bypass (TRICC) study, a randomized, multicenter clinical trial designed to determine safety and efficacy of T3 supplementation in children less than 2 years of age undergoing surgical procedures for congenital heart disease.³

We would, however, counsel some caution in the interpretation of the main results of this study mainly for two reasons:

First, to our knowledge many centers worldwide would use in these patients peritoneal dialysis (PD) in the early postoperative phase to minimize the effects of systemic inflammatory response and achieve adequate fluid balance.⁴ Usually peritoneal catheters are placed at the end of the operation to drain excess fluid as a routine institutional procedure, and PD is started after admission to the intensive care unit either on a prophylactic basis or after oliguria, anuria, fluid overload, hyperkalemia, or acidosis. PD is able to achieve adequate negative fluid balance in nearly all instances and treat other underlying causes simultaneously. Thus the positive achievement of negative fluid balance at day 2 versus day 2.5 may be clinically negligible under most given circumstances.

Second, the authors quote the effect of the T3 infusion on the cardiac output, systolic blood pressure and, herewith, indirectly on the requirements of catecholamines. Unfortunately, no information is provided on the absolute use of vasodilators (such as phenoxymethylamine, sodium nitroprusside, and milrinone), nor is there differentiation in the catecholamine use with regard to dopamine. It is widely known that dopamine has a direct negative effect on thyroid function and thyroid hormone levels in infants and children.⁵ Other factors such as the use of amiodarone should be discussed too. Finally, the overall dropout rate of cardiac output measurements in both groups (14/42) seems relatively high and unbalanced between the two small study groups. This additional information seems crucial to enable a more objective assessment of the study.

Despite these limitations, we believe that the study by Mackie and associates adds valuable information to the controversial dispute on the efficiency of T3 as