**Systemic Vascular Resistance Should Be Banned**

**To the Editor:**

In an article published in a recent issue of *CHEST* (June 2014), Marik1 mentioned, “In patients with septic shock who are fluid responders (an increase in cardiac output with fluid boluses), vasodilatation with a fall in systemic vascular resistance has been observed…. Hence, although the cardiac output increases, vasodilatation occurs.” I believe that this statement is erroneous because there is no way to measure vasodilatation quantitatively. The decrease in systemic vascular resistance (SVR) is simply due to mathematical coupling: \( \text{SVR} = \frac{\text{mean arterial pressure} - \text{central venous pressure}}{\text{cardiac output}} \). According to this equation, SVR must decrease if the cardiac output increases, but in the patient, this does not have to be the case and could lead to dangerous errors in patient management. This error in thinking is made time and again in publications and clinical practice. I believe that SVR is meaningless in clinical practice because it simply does not indicate whether the patient is vasodilated. It is a term derived from Ohm’s law on electrical circuits and has no place in the treatment of patients.

Michael Rodgers, MD
Groningen, The Netherlands

**AFFILIATIONS:** From the Department of Critical Care, University of Groningen, Groningen, The Netherlands.

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**CORRESPONDENCE TO:** Michael Rodgers, MD, Department of Critical Care, University of Groningen, Hanzeplein 1, Groningen 9713GZ, The Netherlands; e-mail: m.g.g.rodgers@umcg.nl

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**References**


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**Response**

**To the Editor:**

I thank Dr Rodgers for his thoughtful comments. I agree that clinicians should treat calculated variables, be these related to acid-base status or hemodynamics, with a healthy degree of skepticism. However, although heart rate, mean arterial pressure, and cardiac output are the primary hemodynamic variables that should concern the intensivist, systemic vascular resistance (SVR) has a theoretical construct that may be useful. For example, if an intervention increases the cardiac output without a concomitant increase in mean arterial pressure, this is best explained by a fall in SVR. Among the articles cited in my review article,1 Pierrakos et al demonstrated a fall in SVR in patients who were volume responders, but it did not fall in the nonresponders. This is best explained by vasodilatation in the fluid responders. Similarly, using esophageal ultrasound, Monnet et al measured the cross-sectional area of the aorta before and after a fluid challenge and demonstrated an increase in area after volume expansion in the fluid responders but not in the fluid nonresponders. These clinical studies support the concept that fluid loading may have detrimental effects in both fluid responders and fluid nonresponders. Furthermore, to answer the question, “Should the term ‘systemic vascular resistance’ be banned,” I would say, no!

Paul E. Marik, MD
Norfolk, VA

**AFFILIATIONS:** From the Division of Pulmonary and Critical Care Medicine, Eastern Virginia Medical School.

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**CORRESPONDENCE TO:** Paul E. Marik, MD, Virginia Medical School, 825 Fairfax Ave, Ste 410, Norfolk, VA 23507; e-mail: marikpe@evms.edu

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