

Response

To the Editor:

We thank Dr Das for his interest in our case series published in CHEST (May 2010).1 In his letter, Dr Das emphasizes some of the key clinical data presented in Table 1 of our report on six patients with both pandemic 2009 influenza A(H1N1) (A[H1N1]) and cardiac dysfunction. We agree that other conditions, including but not limited to sepsis, multiple organ dysfunction syndrome, and peripartum cardiomyopathy, may have contributed to the cardiac dysfunction observed in our case series. Limited by the nature of a small case series, we were careful not to assign a causal relationship to the observed association between A(H1N1) and cardiac dysfunction. Regarding Dr Das’s proposal of right-sided heart dysfunction contributing to left-sided heart dysfunction, transthoracic echocardiographic assessment of right-sided heart function was normal in all of our cases except the patient in case 6, who had moderate right ventricular dysfunction. We agree that in hospitalized patients it is difficult to distinguish whether cardiac dysfunction is attributable to direct viral mediated myocarditis, systemic inflammatory response, or other comorbidities. However, we believe it prudent for clinicians to be aware of a high prevalence of cardiomyopathy associated with A(H1N1) in hospitalized patients.

Subsequent to submission of our case series, several reports have emerged suggesting a direct effect of A(H1N1) on the myocardium. For example, Puzelli et al2 described an 11-year-old child with A(H1N1) in Italy who died of myocarditis and had A(H1N1) isolated from both myocardial tissue and pericardial fluid, providing direct evidence for A(H1N1) effects on myocardial cells. Histologic confirmation of myocardial infiltration with A(H1N1) was obtained in a 5-year-old girl whose death was documented in a series of four cases from a single hospital in California over just a 30-day time period.3 Haessler et al4 reported a case of a previously healthy 24-year-old woman with A(H1N1) and left ventricular dysfunction that improved on serial echocardiograms, consistent with our observations. Beyond traditional echocardiographic assessment, tissue Doppler measurements enabled detection of subclinical cardiac dysfunction in patients from Turkey hospitalized with A(H1N1).5 Furthermore, a report from Japan identified that myocarditis associated with A(H1N1) seemed more common than observed with prior seasonal influenza outbreaks.6

We agree that it is difficult to causally relate cardiac dysfunction to a specific etiology in critically ill patients within a small case series. However, it is our belief that physicians managing patients with A(H1N1) should be aware that transient cardiac dysfunction can occur and may be related to viral myocarditis.

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Femoral Venous Oxygen Saturation May Still Be a Valuable Tool

To the Editor:

We read with great interest the study reported by Davison et al1 in a recent issue of CHEST (July 2010). We completely agree with their hypothesis and part of their conclusion: Femoral venous oxygen saturation (Svo2) cannot always be used as a substitute for central mixed Svo2 in patients who need critical care. However, we think that femoral Svo2 can be used to guide resuscitation. It has been established that regional differences in oxygen consumption account for the regional differences in Svo2;2 hence, Svo2 measurements in the superior vena cava (SVC), inferior vena cava (IVC), and pulmonary artery (PA) can diverge significantly.1,2 Cerebral and cardiac consumption account largely for the Svo2 in the SVC and PA, whereas IVC Svo2 reflects mainly liver, kidney, gut, and skin consumption.3 Thus, the oxygen content in the IVC reflects only a part of the total oxygenation of a patient who is critically ill, but the clinical difference with the Svo2 in the SVC is lessened if the trend in femoral Svo2 is used, rather than the absolute value. Trends can be extremely helpful

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in the treatment of the patient who is critically ill. Moreover, in these patients, since the resting oxygen demands cannot be met, blood flow will be reduced through low-extraction tissues, such as the liver and gut, to be rerouted to essential tissues such as the brain; in this situation, the IVC SvO₂ will reliably show an early decrease, indicating tissue hypoxia. The study by Davison et al demonstrates elegantly the discrepancy between SvO₂ measurements in the upper and lower body in patients who are critically ill. However, considering the low margin between the mean SvO₂ levels in patients who are critically ill. Moreover, the utility of using trends rather than absolute values in the individual patient, IVC SvO₂ is still clinically useful, though numerically different, and should not be discarded from the critical care armamentarium.

Response

To the Editor:

We thank Drs El-Rassi and Yazigi for their thoughtful comments regarding femoral-based central venous oxygen saturation (SvO₂) and its potential use as a surrogate marker for central SvO₂ to help guide resuscitation. Drs El-Rassi and Yazigi point out that the absolute margin of difference between central SvO₂ and femoral SvO₂ reported in our study is small (73.1% ± 11.6% vs 69.1% ± 12.9%, respectively; P = .002). Although the absolute difference is small, when considering the SD (SD ± 11.6%) and large limits of agreement (18.4%–26.4%), the range of discrepancy is significant and clinically relevant.

As stated in our article, according to these findings, a central SvO₂ of 70% corresponds to a femoral SvO₂ range of 58.8% to 81.2%. A value of 58.8% would prompt the physician to optimize oxygen delivery by means of volume resuscitation, blood transfusion, or the initiation of inotropic agents. Yet, a value of 81.2% alone would not generate the same intervention. Thus, we believe that the femoral SvO₂ cannot reliably be used to make clinical decisions or guide resuscitation. SvO₂ values differ among organ systems because each organ extracts variable amounts of oxygen, particularly in states of physiologic stress. Absolute values of venous oxygenation, therefore, depend greatly on the site of measurement. It has been well established that the mixed SvO₂ value obtained from the distal port of a pulmonary artery catheter reflects the venous return from both the upper and lower portions of the body and, therefore, is a marker of global tissue hypoxia. However, it is important to note that the standard femoral venous catheters are 20 cm in length. In the average-sized adult, the tip, and hence the venous sampling, is within the iliac vein and not in the inferior vena cava. Thus, SvO₂ from the intraabdominal organs, including the liver, kidney, and splanchnic regions, is not necessarily sampled. A low femoral SvO₂, therefore, may not necessarily reflect a redistribution of blood flow to indicate that global tissue hypoperfusion is present.

Future studies examining trends of femoral SvO₂ particularly in patients in the early stages of shock, may produce more promising results, and we intend to conduct the same study in patients with shock. Based on our current study, we believe that femoral SvO₂ cannot be used to reliably guide resuscitation. The topic of differences in regional blood flow and oxygen delivery is an interesting one and deserves further evaluation.

Davison et al also point out that the correlation coefficient obtained from the distal port of a pulmonary artery catheter demonstrates elegantly the discrepancy between SvO₂ measurements regarding femoral-based central venous oxygen saturation.

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Survival in Pulmonary Hypertension Registries

The Importance of Incident Cases

To the Editor:

We read with interest the characteristics and survival data of patients with connective tissue disease-associated pulmonary

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