2. Stroke volume index = $SVI = \frac{CI}{HR} \times 1000$ (ml/beat/m$^2$).

3. Left and right ventricular stroke work index:

$$LVSWI = SVI \times (\overline{BP} - PCWP) \times 0.0136 \text{ (g-m/m}^2\text{)};$$

$$RVSWI = SVI \times (\overline{PA} - CVP) \times 0.036 \text{ (g-m/m}^2\text{)}.$$

4. Left and right ventricular end-diastolic volume index:

$$LVEDVI = \frac{SVI}{LVEF} \text{ (ml/m}^2\text{)};$$

$$RVEDVI = \frac{SVI}{RVEF} \text{ (ml/m}^2\text{)}.$$

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## Discussion

DR. LOUIS R. M. DEL GUERCIO (Valhalla, New York): As Dr. Greenfield pointed out, the preload factor in the original Starling-Frank hypothesis had nothing to do with pressure. It was a volume load, and the stretch of the muscle fibers then was related to the subsequent work during systole.

But we compromise, and as biomedical engineers and physiologists in clinical settings, we have a tendency to measure the things that we *can* rather than the things that we *should;* and this is why, over the past many years, we have been measuring filling pressures, first the central venous side, and subsequently, with the advent of the Swan-Ganz catheter, the left side of the heart.

We have noted that the right side of the heart is particularly vulnerable to increases in pressure work. It handles increased volume loads very handily, but in such situations as pulmonary embolism or acute respiratory distress syndromes, the right ventricle is more vulnerable than the left, particularly in young individuals.

We have tended to concentrate on what is happening in the left side of the heart, primarily because we have used the Swan-Ganz catheter for monitoring elderly patients, patients with known arteriosclerotic cardiovascular disease; but in the acute trauma setting and in the acute setting related to massive resuscitation in young people, it is the right side of the heart, as has been shown by Dr. Greenfield, that is very likely to fail. This may be related to the geometry of the ventricle, as he has demonstrated.

We too have found this phenomenon, and are searching for techniques to measure ejection fractions, which, after all, goes all the way back to the original Starling-Frank hypothesis. Our only hope is that modern technology will enable us to achieve this sort of monitoring with a technique that can be easily applied to the bedside in the clinical shock setting.

DR. WATTS R. WEBB (New Orleans, Louisiana): Our own work, both in hypervolemia and in septic shock, both clinically and experimentally, demonstrates, we feel, that there is virtually always a marked increase in pulmonary artery pressure, and, similarly, in pulmonary artery resistance, which was alluded to by Dr. Greenfield here. It is not related to the wedge pressure at all, but I think that the flow and the amount of resistance are the things that are the most important.

I think that the increased volume, which is here characterized as a dysfunction, may be just a shift to the right of the Frank-Starling curve, thereby giving increasing efficiency. The fact that the central venous pressure in most of these patients did not rise would suggest to me that this, actually, was not an indication of true failure of the right ventricle.

I think we have looked too long at filling pressures as the measure of resuscitation of our patients who are in shock for any reason. We should instead turn to resuscitation to a relatively normal blood volume, to a relatively normal cardiac output, with normal, or low normal, pulmonary artery pressures and pulmonary artery resistance, particularly, maintaining low pulmonary artery and systemic resistances. In most of the patients, this is going to require the use of some vasodilator, such as nitroprusside, which will vasodilate the peripheral as well as the pulmonary vasculature. We find in most of our patients, and certainly our experimental dogs, that the pulmonary resistance is going to rise two- to four-fold. And I believe that Dr. Greenfield's work here demonstrated that those who were in difficulty were those who had a rise in pulmonary resistance.

So my question would be: Is this really a dysfunction, or is this the normal Frank-Starling response to the increased afterload that is imposed on the right ventricle?

DR. JAMES V. MALONEY, JR. (Los Angeles, California): Dr. Hoffman and Dr. Greenfield and their associates have identified something that I'm sure all of us must have seen, but not recognized. I think these patients have a standard type myocardial infarction that is normally seen on the left side of the heart, by the following mechanism.

The left ventricle, as you recall, during systole normally has a coronary driving pressure of 120 in the aortic root and in the coronary arteries. Therefore, no flow can go to the left ventricular myocardium during systole. During diastole, when the aortic pressure is 80, and end diastolic pressure in the ventricle is 10, all the coronary flow to the left ventricle occurs.

The right ventricle is quite different. During systole, the pressure in the coronary artery is 120, and the pressure in the right ventricle normally is 30; so the right ventricle gets, probably, half its flow during systole, and in addition, it gets the rest of its flow during diastole, when the aortic pressure is 80, and right ventricular pressure, for example, is 5.

However, in shock, quite a different set of circumstances occurs, particularly if one has pulmonary hypertension. The right ventricle cannot get adequate blood supply unless blood is delivered during systole. Any newborn infant who has isolated pulmonary valvular stenosis, who has a Blalock shunt performed—essentially, all of them die, and the reason is that the high pressure persists in the right ventricle, and when you do a shunt, you reduce diastolic pressure, and therefore no coronary flow occurs.

As far as the clinical syndrome goes, when we used to damage hearts for an hour or two during cardiac bypass, some of the patients died immediately, as Dr. Greenfield's patients did, and some, when studied remotely, later, have decreased ejection fraction and high diastolic filling pressures. And it was with increasing excitement as this paper unfolded that I saw that exactly the same thing occurs here. And then the *piece de resistance* was when Dr. Greenfield said that the one correlate they found in this syndrome was that if the pulmonary vascular resistance index is high, it is associated with deterioration. Of course, what that means is that the pulmonary vascular resistance index is high, the pressure on the right side of the heart is high, and therefore the pressure in the aortic route is not high enough during that period of shock to supply the right ventricle with blood. And what we are seeing here, I believe, is right ventricular myocardial infarction in the presence of normal coronary arteries, which is a condition which we have recognized in the cardiac field in the last 4 or 5 years as occurring in the left side of the heart as well.

I am astonished that all of us have seen this syndrome so far in the past and have failed to recognize it. And I suggest this as an alternative explanation.

DR. RICHARD M. PETERS (San Diego, California): Dr. Greenfield suggests from his information that, for some reason, the compliance of these right ventricles is varying because he has no correlation between filling pressure and end diastolic volume. Does he have any explanation for why the ventricle has such inconstant right ventricular compliance?

DR. LAZAR J. GREENFIELD (Closing discussion): Dr. Del Guercio has kept us aware of the importance of bedside measurement of hemodynamics in patients for a long time. I think that we have the prospect of being able to provide this information more easily in the future. Currently, we and others are working, on the analysis of the thermal dilution curve in a way that will allow us to calculate ejection franction and end diastolic volume.

Dr. Webb's comments were in relationship to whether or not failure is actually occurring in these patients, and we do have some further correlations with cardiac index but the real test will be whether or not we can by modifying the right ventricular dimensions and performance, provide a better outcome for the patients.

I think the comments regarding vasodilatation may well be appropriate, but there is an obvious danger in the use of dilator agents for the very reason that Dr. Maloney mentioned; and that is that a reduction in coronary perfusion pressure with reduced systemic pressure might well be deleterious for the right ventricle. So we will have to investigate other mechanisms for trying to normalize right-sided performance.

Dr. Maloney's comments are welcome, and we certainly agree that right ventricular ischemia is probably responsible for some of the deterioration in these patients. It has been well demonstrated experimentally that the right ventricle can tolerate increasing loads such as progressive pulmonary artery constriction—if coronary perfusion is improved. And, in fact, that is a major determinant of the right ventricle's ability to tolerate this workload.

Dr. Peters' comments regarding compliance of the ventricle are appropriate and indeed, compliance is changing in these ventricles. Perhaps it is due to changing pulmonary vascular resistance but we don't know, and that is an important question for us to answer in the future.