increased slightly. This suggests that the heart was being compressed by the lungs and/or a zone 2 condition had been created. A definitive solution to these problems is not readily available.

The possible influence of ventricular interdependence on LV performance cannot be ignored. It is entirely possible that right ventricular volume is decreased with binding, particularly during inspiration. This would tend to increase LV compliance and result in small changes in pressure which could reflect large changes in left ventricular volume. More importantly, the effect of such changes in septal geometry on systolic pump function in the compromised left ventricle remains a mystery.

Further complicating the issue of left ventricular volume measurement are the results of recent studies which demonstrate asymmetrical regional left ventricular shape changes during a respiratory cycle. These findings make it clear that either a nongeometric technique (such as MUGA) or measurement of at least three orthogonal axes are required to determine LV volume. Thus, both M-mode and 2-D echocardiograms, or single plane angiograms may generate highly misleading volume determinations, since the unmeasured dimension(s) may be changing in a direction opposite those measured.

The clinical investigator who manipulates intrathoracic pressure during ventilation (e.g., PEEP) will, in the future, have to determine not only total cardiac output, but perhaps as importantly, its regional distribution. With thoraco-abdominal binding, one would predict preferential flow of left ventricular output cephalad, with a relative diminution of flow to the abdominal compartment. Thus, the paradoxic situation can exist in which an increase in abdominal pressure can actually decrease total LV output by increasing the impedance in the abdominal aorta while simultaneously increasing flow to the arterial beds cephalad. We must therefore consider the longer term effects of such ventilatory manipulations in reducing hepatic, splanchic and renal perfusion or adversely overcorrecting cerebral blood flow in patients susceptible to cerebral hemorrhage.

In conclusion, the development of techniques in which lung volume and intrathoracic pressure are manipulated to improve both global and regional cardiac output, ventilator-assisted myocardial performance (VAMP; if I may), is not likely to be a panacea, but holds the possibility of adding one more finger in the dike when time is required for definitive therapy or gradual healing. Only many more carefully done bedside studies will determine if instead of deciding "to PEEP" Mr. Jones with his failing left ventricle, we may decide to "VAMP" him.

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Crystalloid or Blood Cardioplegia with Cardiac Surgery

By keeping the heart profoundly cooled and completely arrested, the cold cardioplegic technique of myocardial preservation allows the surgeon to safely use a single period of ischemic arrest to accomplish most cardiac operations. A question of major clinical importance is whether a further increment of protection can be gained by intermittently delivering oxygen to the myocardium while it is arrested using the technique of blood cardioplegia as described in the article in the April issue of Chest by Catinella et al. Their findings are supported by a number of studies that also have suggested that blood cardioplegia might better protect the heart during ischemic arrest than do various techniques using crystalloid cardioplegia. We recently completed a survey to determine the frequencies of various perfusion techniques and received responses from over 600 clinical perfusionists in the United States and Canada representing an annual
case load of over 140,000 cases. We found that two-thirds of the respondents still used only crystalloid cardioplegia, while 15 percent used only blood cardioplegia and 17 percent used both. Clearly, most surgeons are not as yet convinced that the data supporting the superiority of blood cardioplegia warrants a change in their clinical practice.

The technique using crystalloid potassium cardioplegia delivered at 2° to 4°C with reinfusion intermittently about every 30 minutes combined with moderate systemic hypothermia can consistently keep the myocardial temperature in the range of 8° to 15°C during the ischemic period even with all the manipulations necessary to accomplish the usual cardiac operation. This method has provided excellent clinical results, but those of us who continue to use it must concede that there is still room for improvement. Although it is unusual to see myocardial necrosis or even transient significant myocardial dysfunction after even the most complex elective cardiac operations using this method, these problems still do occur, but mostly in patients who have had poor hemodynamics prior to bypass who enter the period of ischemic arrest with metabolically compromised myocardium. It is for this group of patients that we must ask whether an added increment of protection, such as that suggested by the study by Catinella et al, for the technique of blood cardioplegia, might provide sufficient improvement in myocardial protection to improve postoperative hemodynamic performance and further decrease mortality.

Since there has not been a conclusive clinical study that has answered the question of whether blood cardioplegia is indeed superior to the protection achieved with the best methods using crystalloid cardioplegia, we must today rely heavily upon laboratory studies such as that by Catinella et al to guide our clinical practice. They have studied one variable, namely: myocardial tissue adenosine triphosphate (ATP) levels during and after a three-hour ischemic period. Adenosine triphosphate levels immediately after ischemia and especially after recovery with reperfusion are considered to be a good indicator of the metabolic status of the myocardial cell and have correlated well with myocardial function and hemodynamic performance after ischemic arrest. Though determination of hemodynamic recovery would have added strength to their conclusion, the study still represents another important piece of data to support the superiority of the blood cardioplegia technique.

There are two important concerns about blood cardioplegia that probably account for the fact that it is not universally accepted for clinical practice. Because of the nature of the hemoglobin-oxygen dissociation curve with hypothermia, oxygen delivery to myocardial tissue is increasingly limited as the blood cardioplegic solution is cooled to within the range necessary to achieve profound myocardial hypothermia. There are good data to suggest that myocardial tissue oxygenation is not substantially improved when the myocardium is perfused with blood cooled to 10°C or lower. Most studies that have compared blood with crystalloid cardioplegia have, in fact, infused the blood cardioplegia at temperatures between 10° and 20°C. In the study by Catinella et al, each of the arrest solutions was delivered at 12° to 13°C and the myocardium was maintained between 10° and 15°C with topical hypothermia. In the laboratory, topical hypothermia can be extremely effective in maintaining profound hypothermia. However, in the clinical setting, especially during coronary artery bypass surgery, topical hypothermia cannot be nearly this effective and one must rely more on cooling with cardioplegic reinfusion to maintain deep hypothermia. The choice then is to deliver a crystalloid solution at less than 4°C and be able to consistently maintain the lowest myocardial temperatures or to compromise on myocardial cooling and deliver blood cardioplegia at a temperature in the range of 12° to 20°C so that one can get the benefit of oxygen delivery to the myocardium. Many surgeons probably prefer the security of maintaining the lowest possible myocardial temperature to the possible benefit of intermittent oxygenation.

The other concern is that whereas with crystalloid cardioplegia there is complete control over the contents of the solution delivered to the myocardium, one does not have this control with blood cardioplegia and may at times unnecessarily subject the ischemic myocardium to the harmful effects of high catecholamine levels as we know are sometimes present during carduopulmonary bypass. Although a variable degree of washout by non-coronary collaterals does occur during aortic crossclamping, thus limiting the importance of this difference, if high catecholamine or other vascular substance levels in blood cardioplegia represent a significant factor in only the occasional patient, its effect may not be seen in a limited laboratory study and might only be evident in a large clinical study.

Another alternative that has been suggested is to use oxygenated crystalloid cardioplegia which will release nearly as much oxygen to the myocardium as will blood cardioplegia at the lowest temperatures. This is clearly a compromise, but does permit one to control the makeup of the solution delivered to the myocardium and also to maximally cool the heart while delivering limited amounts of oxygen intermittently during the ischemic period. However, as with blood cardioplegia, there is no solid clinical evidence that this is superior to methods of crystalloid cardioplegia commonly used today. Unfortunately, since any of these methods provide consistently good results in the vast majority of cases, we must look forward to further supportive evidence from good laboratory studies and
hopefully from well designed clinical studies to determine finally which of these techniques is truly the best.

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