Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Effects of Amrinone in Patients Recovering from Cardiac Surgery

To the Editor:

I would like to offer a few comments on the article by Prielipp et al., which appeared in the April 1991 issue of Chest. I found the authors’ conclusions as interesting for what they did not say as for what they did ascertain from their published data.

First, I think the authors’ statement that “amrinone increased CI [cardiac index] in a dose-dependent manner” is misleading in tone and connotation. On examination of the data, it can be seen that amrinone did significantly improve CI, but it did so through an increase in heart rate. Average stroke index as calculated from their hemodynamic tables shows an increase from 32 ml/sq m to 34 ml/sq m in the low-dose group and from 31 ml/sq m to 34 ml/sq m in the high-dose group. Although I cannot say from the data shown, I would be greatly surprised if these were significant changes. The hoped-for utility of amrinone is its ability to augment CI through an increase in stroke index due to both its unloading and its direct contractility effects, thereby improving ventricular mechanics with a minimum cost in additional myocardial oxygen consumption. The specific hemodynamic effects of amrinone in the patients in this study might actually be considered by many clinicians as an undesirable effect. I think this should be pointed out to qualify the above-mentioned statement by the authors.

Amrinone enjoys the reputation of having the least chronotropic effect among its commonly used catecholamine brethren, dopamine and dobutamine. The curious response observed in this study may be due to what I suspect was a state of relative volume depletiion reflected in the pulmonary artery occlusion pressure (PAOP) data. Perhaps a different kind of response would have been observed in patients with PAOP more in the range of 14 to 18 mm Hg.

Second, I wish to comment on the authors’ conclusion concerning the utility of mixed venous gas monitoring. I completely agree with the authors’ points, but again, I must object to the tone and connotation of what is said rather than the exact text. The simplicity of the Fick equation belies a complex of physiologic principles that directly relate to the issues addressed by their article. The Fick principle shows that the true utility in monitoring mixed venous blood is the resultant capability to track oxygen extraction via the arteriovenous oxygen content difference (Ca-vO2). It is this parameter that correlates with the interplay between oxygen delivery and oxygen utilization on the systemic level. The value of continuous (or discontinuous) mixed venous oxygen saturation monitoring is the access it provides the clinician to data essential to the monitoring of this physiologic variable. In this article, the average increase in oxygen delivery in the high-dose group correlated with a drop in average Ca-vO2 from 6.9 volume percent to 5.9 volume percent, a result consistent with theoretical expectations. The Ca-vO2 would have been a more meaningful variable to correlate with the physiologic effect of amrinone, and thus might have confirmed the utility of mixed venous oxygen saturation monitoring.

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Reference


To the Editor:

We appreciate the comments of Dr Yannios concerning our article. As noted in our data, CI increased in a dose-dependent fashion after amrinone administration. This increase in CI was a result of a significant increase in heart rate and the trend for an increase in stroke volume. This hemodynamic profile clearly reflects drug effect, loading conditions, intrinsic sympathetic stimulation, resting cardiac function, and postoperative cardiac recovery of ventricular function during our study. As Dr Yannios correctly points out, this profile would likely have been significantly different had cardiac preload (and other loading conditions) been simultaneously manipulated during amrinone infusion, but we felt that such manipulation would have made subsequent interpretation of the data even more complicated with respect to sorting out primary drug (amrinone) effect. We would agree that caution should be exercised in extending the hemodynamic findings from this study patient population (ie, stable postoperative patients with adequate cardiac function) to other groups of patients actually requiring cardiac drug therapy (eg, ventricular failure, stunned myocardium). We are, in fact, presently studying a group of patients with depressed ventricular function immediately following separation from cardiopulmonary bypass.

Examination of the two drugs mentioned by Dr Yannios (dopamine and dobutamine) illustrates the potential variability of hemodynamic profiles when drugs are studied in these different settings. In stable postcardiac surgery patients in the ICU with adequate cardiac function, DiSesa et al found that neither dopamine nor dobutamine produced a significant increase in stroke volume, but both agents significantly increased CI by increasing heart rate. In contrast, in cardiac surgery patients studied in the operating room immediately following cardiopulmonary bypass, Steen et al found significant increases in CI and stroke volume index with both dopamine and dobutamine. In that study, left atrial pressure was maintained constant by additional transfusion as needed. Clearly, caution should be exercised in extrapolating information gathered in one setting and applying it to another without prospective confirmation.

Mixed venous oximetry is advocated as a monitor in critically ill patients and current technology allows continuous mixed venous saturation monitoring, albeit at additional cost. Several studies cited in our article both support and refute its utility in the ICU setting. As Dr Yannios correctly states, many additional measure-
ments are available from the pulmonary artery catheter, which may be useful in titration of inotropic therapy. These parameters, including oxygen delivery, oxygen consumption, and C(a-V)O₂, have been recommended to optimize myocardial performance, systemic perfusion, and survival in critically ill patients. Nonetheless, utilization of only mixed venous saturation (available as the “online” continuous monitor via oximetry technology) might be misleading during acute inotropic titration in cardiac surgery patients; correct interpretation requires consideration of simultaneous influence on cardiac output and intrapulmonary shunt.

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REFERENCES

"Mask" Ventilation Doesn't Have to Be through the Nose

To the Editor:

My colleague Dr Augusta Alba has used intermittent positive-pressure ventilation (IPPV) delivered via the mouth for patients with neuromuscular ventilatory insufficiency since 1957 and has used this technique for ventilatory support for up to 24 h/d in many such patients with little or no measurable vital capacity since 1964. I have been prescribing mouth IPPV since 1977, and I have been using nasal IPPV for patients with acute or chronic ventilatory insufficiency in the United States since 1980.

In the March 1991 issue of Chest, Marino described the use of nasal IPPV by patients with acute respiratory failure due to predominantly intrinsic lung disease. Dr Alba and I are both delighted to see a new application of this technique. It was not clear from reading Dr Marinos work, however, whether nasal IPPV was provided solely with the patients awake, when optimal efficacy and respiratory muscle rest would have been expected, or during periods of sleep as well. We have found that for certain patients with neuromuscular disorders, several hours of ventilatory aid in the evenings before sleep can relieve fatigue and improve unaided blood gas values until the point at which chronic hypoventilation becomes so severe as to warrant nocturnal IPPV as well. These techniques can both substitute for and provide rest for respiratory muscles and may be useful alternatives to questionably efficacious negative-pressure aids for patients with obstructive pulmonary disease.

It was unclear whether Dr Marinos patients who failed to benefit from IPPV via continuous positive airway pressure (CPAP) masks failed strictly because of mask discomfort (a contributing factor in at least four patients), inadequate mask fit with leakage, excessive oral leakage during sleep, or other reasons. Apparently only one style of commercially available CPAP mask was used. Nasogastric tubes, which are commonly present in the intensive care patient, can render CPAP/mask nasal IPPV intolerable and also ineffective by impairing fit and preventing sealing off of the oral cavity by the soft palate. Custom molding of nasal interfaces, an option since 1987 and now available commercially, and mouth IPPV are options for some of these patients. A custom-molded nasal interface, the SEFAM mask (Lifecare, Lafayette, Colo) can be fabricated in 15 min. It, and other custom-made pieces, can maintain a better seal at higher pressures with greater comfort.

One of Dr Marinos patients failed to improve because he had a "severely congested nose and could not tolerate a full face mask." The author stated that "positive pressure ventilation has been administered via the lip-seal device, but this is rather uncomfortable. The reference mistakenly cited to support this statement had no information about a lip-seal device." He also stated that he could achieve higher pressures using nasal IPPV rather than mouth IPPV and that for one patient a chest shell was preferred. However, this information implies that when attempting mouth IPPV his patient never learned how to use his soft palate to prevent excessive air leakage out of his nose. These points illustrate the fact that mouth IPPV is commonly misunderstood. Perhaps a lesson can be gleaned from the management of neuromuscular patients. Commercially available CPAP masks can rarely be used to provide ventilation at pressures exceeding 25 cm H₂O without significant air leakage and pressure discomfort. Although somewhat greater pressures can be delivered via molded interfaces, pressures of 40 cm H₂O or greater can be readily obtained via a mouthpiece with no discomfort at all. Since Dr Marinos patients used IPPV predominantly while awake and since they presumably had normal upper extremity function, resort to a lip seal should have been entirely unnecessary. Indeed, we do not even use lip seals for the delivery of mouth IPPV to awake neuromuscular or high-level traumatic quadriplegic patients who have no upper extremity function at all (Fig 1). Yet, for most clinicians, when nasal IPPV, especially nocturnal nasal IPPV, is

FIGURE 1. A traumatic quadriplegic with little autonomous ventilatory capacity who was converted from tracheostomy to mouth IPPV via a small flexed mouthpiece during the day and mouthpiece with lip seal overnight. The tracheostomy site was allowed to close.