operation.

Preston's insinuation that cardiologists and surgeons are recommending aortocoronary bypass surgery for personal economic reasons is one of the most insulting things that I have ever read in a medical journal. Did it ever occur to him that the results he reads about in journals and sees in government-funded hospitals might not be the same as the results obtained by competent cardiologists and experienced skilled surgeons in private practice? There is no comparison between the results that we obtain in our private practice and those I read about in medical journals. Our results are far superior. We recommend aortocoronary bypass surgery because it is best for our patients.

Rodney L. Crislip, M.D., F.C.C.P.
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REFERENCE


To the Editor:

Crislip makes many good points about the limitations of controlled studies. No therapy is uniform (not even propranolol, which is given in varying doses, sometimes inappropriately), resulting in a problem of extrapolating from the results of a study to the general population. Although it is highly unfair and inaccurate to charge that surgeons participating in controlled studies are inferior, if there is reason to believe that better surgery would produce better therapeutic results, the only way to test this possibility is for the alleged better surgeons to do a proper controlled study. Uncontrolled studies have all the faults of controlled studies, in addition to having no fair means of comparison. Only with no valid means of comparison between treated and untreated groups and with no understanding of the need for controls could anyone claim that "our results are far superior," with no supporting evidence. Although surgical expertise is undoubtedly important, a possibly more important determinant of results is the selection of patients, which must be the same for any fair comparison between groups. Is it sufficient that we support our actions with no more evidence than a statement of belief, as does Crislip, or does the public deserve scientific evidence of benefit from our type of therapy?

I certainly do not think that "the only way to knowledge" is through controlled studies, but I do feel strongly that uncontrolled studies can be very misleading. I personally believe that coronary arterial surgery is beneficial for many patients, but I want to avoid the self-deception that can result from our own unsubstantiated beliefs. For every example of specific anatomic or pharmacologic correction of known mechanisms of disease, there are hundreds of treatments that seemed to be physiologically correct but ultimately proved worthless. Recent analyses show that the great majority of surgical innovations in the last ten years have not been an improvement over existing therapy. Our belief alone is not enough. When a controlled study is possible, it is unethical to utilize uncontrolled studies, which are notorious for supporting the beliefs of investigators and which often give misleading results.

In evaluating aortocoronary bypass surgery, we must include the negative reactions as well as the benefits. The skill of the surgeon is part of the therapy. It is the therapy, not the operation, that we must evaluate. Otherwise, "the operation was a success, but the patient died" would be acceptable.

Financial incentives are important in any transaction involving payments. I do not believe that cardiologists or surgeons make final decisions on the basis of personal economics, but financial incentives play a role in drawing physicians into certain types of practices, in the planning of hospitals, and in the utilization of technologies. Is it not proper to ask whether financial incentives are the reason that coronary arterial surgery is performed two to three times as often in private practice as in health maintenance organizations and ten times as often in the United States as in western Europe? The problem is really not the incentive, but the absence of disincentive, in that most of the charges are borne by third parties. Nevertheless, someone must speak for the consumer and must ask whether financial incentives result in excess surgery (or endoscopic examinations or injections of vitamin B₁₂). I do not offer an answer to these questions, but if the medical profession ignores the matter, for whatever reason, it will surely lose the autonomy necessary to maintain control of medical economics within the professional domain.

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Influence of Artificial Ventilation on the Pulmonary Capillary Wedge Pressure

To the Editor:

We were very surprised by the data observed by Davison et al on the influence of artificial ventilation with intermittent positive-pressure ventilation (IPPV) or positive end-expiratory pressure (PEEP) on the pulmonary capillary wedge pressure. We have found that therapy with IPPV influences the pulmonary capillary wedge pressure, sometimes very strongly, in patients with acute respiratory failure. Several authors, including us, have observed that the influence of therapy with PEEP on the pulmonary capillary wedge pressure is even more important.

The modifications of pulmonary capillary wedge pressure induced by therapy with artificial ventilation appear to be logical because the pressure measured is an intravascular pressure that reflects the sum of transmural and extravascular pressures. Therapy with IPPV and especially artificial ventilation with PEEP increase alveolar pressure and modify the value of the pulmonary capillary wedge pressure; thus, the value measured during therapy with artificial ventilation does not reflect the left ventricular filling pressure. Moreover, therapy with artificial ventilation lowers the central blood volume and thus decreases the left ventricular filling pressure. Therefore, with therapy with artificial ventilation, it is hypothetical to deduce the value of the left ventricular filling pressure from the pulmonary capillary wedge pressure measured after a short weaning from the respirator.

These findings are sustained by the opposite influence of therapy with artificial ventilation in edema due to increased permeability of pulmonary capillaries and in hemodynamic acute pulmonary edema. This last phenomenon, observed by Davison et al, is for us very important because therapy with artificial ventilation can artificially reduce the pulmonary capillary wedge pressure to a normal value in hemodynamic acute pulmonary edema. On the contrary, in hypovolemia, therapy with artificial ventilation can raise the
pulmonary capillary wedge pressure to normal by increasing extramural pressure. So far, therapy with IPPV and PEEP usually influences the pulmonary capillary wedge pressure, especially when the central blood volume varies from normal; thus, the interest and limits of the "weaning test" are clearly defined.

In some cases, therapy with artificial ventilation does not change the pulmonary capillary wedge pressure. This results from either a balance between the change of central blood volume and extramural pressure induced by artificial ventilation or the severity of the pulmonary diseases. In the latter case, very high airway resistances or very low pulmonary compliance (or both) can explain why airway pressures are not transmitted to the vessels, at least where the catheter is.

To suppress the influence of artificial ventilation, Davison et al used the value of the pulmonary capillary wedge pressure at the end of expiration. For us, this point of view is illogical, since the central blood volume changes during the respiratory cycle. Therefore, the value of the pulmonary capillary wedge pressure measured at the end of expiration cannot reflect the mean left ventricular filling pressure. With therapy with PEEP, especially at high levels (which were not used by Davison et al), residual pressure influences extramural pressure and, therefore, pulmonary capillary wedge pressure at the expiratory stage.

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REFERENCES


To the Editor:

The discrepancy between the data of Labrousse et al and ours is probably more a reflection of procedural differences than anything else. There are many factors that will affect the measurement of pulmonary wedge pressure in patients who are removed from therapy with mechanical ventilation. If the patient displays spontaneous respiratory efforts, the values will vary considerably, depending on the phase of respiration when the measurement is taken. If therapy with positive-pressure ventilation is discontinued for any length of time, significant shifts in volume will occur and will result in the measurement of pulmonary wedge pressures that probably no longer reflect the hemodynamic circumstances existing during therapy with mechanical ventilation.

We are familiar with the theoretic considerations discussed by Labrousse et al but believe that many of them are not borne out by more recent data. The claim that pulmonary wedge pressure measured at the end of exhalation reflects mean left ventricular filling pressure was not made in our article. Nevertheless, we do recommend that in the patient without spontaneous respiration who is receiving therapy with mechanical ventilation, measurements of pulmonary wedge pressure should be taken at the end of exhalation. The "distortion" induced by mechanical ventilation (with positive end-expiratory pressure up to 10 cm H2O) will, in this setting, be insignificant.

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REFERENCES


Ventricular Tachycardia and the Chest Thump

To the Editor:

The report by Conner et al in Chest proposed that self-administered thumping on the chest should be taught to all patients predisposed to recurrent ventricular tachycardia. I would like to call attention to a potential problem which has yet to be resolved. A nonsynchronized stimulus (such as chest thumping) has the potential to induce ventricular fibrillation. This risk, albeit quite small, may be acceptable in the hospital, where equipment for defibrillation is readily at hand. This is not the case in the home. I pose the question to your readers: Has anyone had personal experience with instances of documented ventricular fibrillation induced by chest thumping? Aside from the report of no instances in 68 patients in one study, the question remains open.

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REFERENCES


To the Editor:

As Dr. Rozanski states, the risk of a self-administered thump on the chest inducing ventricular fibrillation is quite small. We have never seen it occur. In fact, there is little evidence that it does occur in the absence of severe and generalized anoxia. Patients with recurrent ventricular tachycardia most often suffer from unexplained disturbances in conduction, ischemic heart disease, myocarditis, etc. In each instance, the risk of spontaneous degeneration of the arrhythmia to ventricular fibrillation is a real one, and one which we believe warrants the trivial risk of inducing ventricular fibrillation by...