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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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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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 available. 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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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
The Genetics and Semantics of Hypertrophic Cardiomyopathy

To the Editor:

We read with interest the article by Maron et al.1 We would like to commend them for utilizing the term, "hypertrophic cardiomyopathy," in this report, instead of the term, "asymmetric septal hypertrophy," which has now proven rather nonspecific. We were also pleased to note that Maron et al2 now recognize that asymmetric septal hypertrophy is not pathognomonic of hypertrophic cardiomyopathy.

Nevertheless, we are concerned about the use of the term, "disproportionate ventricular septal thickening," which seems to further confuse the semantics. It would appear that there may be some difference between disproportionate ventricular septal thickening and asymmetric septal hypertrophy, particularly in view of the following opening statement in a previous report: "Asymmetric septal hypertrophy (ASH) or hypertrophic cardiomyopathy, is a genetically transmitted disease of cardiac muscle that is characterized by disproportionate thickening of the septum."3(290) This statement was supported by several references from the same group,2-5 yet the difference is not clear, as both terms are defined as a septal-free wall ratio of 1.3 or more.1,3

The finding in this article that disproportionate ventricular septal thickening or asymmetric septal hypertrophy occurs in 6 percent (2/33) of the patients with systemic hypertension is considered by Maron et al2 to reflect a relatively low incidence; however, since hypertension is found in 15 to 30 percent of the US population,6 the number of patients with disproportionate ventricular septal thickening secondary to high blood pressure may exceed the number of patients with asymmetric septal hypertrophy associated with hypertrophic cardiomyopathy. The relative prevalence of "genetic asymmetric septal hypertrophy" would be further diminished if we consider the patients with nongenetic asymmetric septal hypertrophy and coronary arterial disease5 or congenital heart disease.7

Since the initial genetic studies were performed at a time when asymmetric septal hypertrophy was believed to be specific for hypertrophic cardiomyopathy and when the occurrence of this entity with symmetric hypertrophy8,9 was not yet recognized, one wonders whether the opening statement that "hypertrophic cardiomyopathy is a genetically transmitted disease of cardiac muscle that is characterized by asymmetric septal hypertrophy"2(438) is still appropriate? A clarification by Maron et al of their present understanding of the genetics and semantics of hypertrophic cardiomyopathy in the light of their recent data would therefore be very helpful.

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