on the ventilator for more than 25 days, and four of these seven could not be weaned in spite of receiving nutritional support. Hence, the area of potential disagreement between myself and Drs. Teba et al is what to do with the patient who will only be on the respirator for a matter of weeks. Is that period of time too long to go without nutrition? During those weeks, will malnutrition significantly add to the respiratory failure? Will the provision of nutritional support benefit in this regard? Will this benefit justify the risk and cost of the nutritional support? At this time, the answers to all of these questions are unknown; only prospective randomized controlled trials (PRCT) will be able to shed any light.

The literature is filled with retrospective and/or uncontrolled reports extolling the benefits of nutritional support in a variety of disorders. However, when put to the test of PRCT, clinical advantages have been found wanting. Although nutritional parameters (e.g., body weight, serum albumin, or anthropometric criteria) have improved, no benefit in the clinical outcomes relating to morbidity or mortality have usually been seen. Even if small benefits were missed (the "type II error"), the best that can be said for nutritional support at this time is that it may offer some small advantages. These can only be identified and quantitated in large PRCT.

Most ventilator-bound patients are weaned in a matter of days or weeks and can then receive food. Given our past experiences with nutritional support in general and the absence of any good (i.e., PRCT) clinical evidence that it is of use in these respirator patients in particular, it is at least premature to advocate the wide-scale application of "artificial feeding" in their care. As is true for any other type of therapy, nutritional support must be shown, and not just theorized, to be efficacious.

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Management of Patients following Correction of Tetralogy of Fallot and Ventricular Septal Defect

To the Editor:

In a recent review article, Dr. Krongrad discussed the management of patients with intraventricular conduction defects developing after repair of tetralogy of Fallot or ventricular septal defect (Chest 1984; 85:107-10). It was pointed out that the group of patients who developed late complete heart block includes a disproportionate number whose standard electrocardiogram shows the pattern of right bundle branch block, left axis deviation and PR prolongation. However, if clinicians rely on the presence of these three features of the standard electrocardiogram to identify patients at risk for late complete heart block, then some high risk patients would be overlooked. Presumably the PR prolongation is the result of a prolonged HV interval, indicative of conduction abnormality distal to the His bundle and of increased likelihood of late surgical block; the usual site of surgical complete heart block, early or late, is distal to the His bundle. Since prolonged HV interval may occur with a normal PR interval, the PR interval may be less definitive than the HV interval in identifying the high risk group. Dr. Krongrad has previously reported the potential usefulness of measuring HV interval, which can now be done noninvasively. I believe a better appraisal of the risk of late postoperative complete heart block would be obtained if such electrophysiologic investigation was included in the management at least of patients with the pattern of right bundle branch block and left axis deviation whether or not the PR interval is prolonged.

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To the Editor:

Dr. Serratto suggests that some patients with normal P-R interval may have underlying—"concealed"—A-V conduction defects with H-V prolongation. As shown by her and her coworkers, this is indeed possible. The issue is not whether such an electrophysiologic mechanism may exist, but rather how often does it exist, what are the implications of its presence on prognosis, and what can we do about it to prevent a fatal outcome?

It is our experience that although such a mechanism is possible, it occurs quite rarely in surgical patients (at least in our institution). It is likely that such "concealed" H-V prolongation may have prognostic implication, but as mentioned in the above paper, only one of 204 similar patients reviewed in the literature developed complete heart block and four died suddenly (about 2 percent). Although the patient who developed complete heart block clearly has A-V conduction defect, the four sudden deaths may have been caused by ventricular irritability. Ventricular irritability is an electrophysiologic mechanism and a risk factor discussed later in the above manuscript and is of significant concern among pediatric cardiologists. Thus, the overall risk for clearly developing severe A-V conduction defects seems to be rather small.

Finally, even assuming such a concealed H-V interval prolongation would be detected by electrophysiologic studies, what should be done for the occasional patient with these findings? I doubt that Dr. Serrato would recommend any therapeutic measures just for H-V prolongation?

Therefore, under these circumstances of rather infrequent occurrence, relatively low prognostic risk, and absence of a reasonable therapeutic intervention, I feel hard-pressed to recommend electrophysiologic studies for patients with postoperative right bundle branch block, left superior axis, and normal P-R interval just to look for the occasional concealed H-V prolongation.

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