and gastric cultures” by Spilker and colleagues (CHEST, July 1996), in which they did not find a difference in gastric pH or gastric colonization in ventilated patients who were switched from continuous to intermittent enteral feeding. We conducted a similar study, but noted significantly different results.2

Our study (Skiest et al)3 was a prospective, randomized controlled trial involving 16 patients receiving mechanical ventilation, who were randomized to continuous or intermittent enteral feeding (16-h continuous, followed by an 8-h fast) for 5 days. Gastric aspirates were cultured quantitatively twice daily, and gastric pH was measured twice daily. The mean AM (postfasting) gastric pH was significantly lower in the intermittent cohort (3.05 vs 4.69, p=0.0008). We also found a correlation of higher gastric pH with colonization. We found lower rates of gastric colonization in the intermittent group on days 4 (0.98 vs 4.23 colony forming units [cfu]/mL, p=0.01) and 5 (0.92 vs 4.92 log cfu/mL, p=0.001).

There are several possible explanations for the differences in our results from those of Spilker and colleagues. First, our study design differed from theirs in several respects. Our study design was prospective, randomized, and included a control group, whereas in the Spilker et al study, patients served as their own controls. Second, concurrent H2 blockers were given to six of their 13 patients, which may have minimized the effect of intermittent feeding in reducing gastric pH and colonization. In our study, antacids, H2 blockers, and proton pump inhibitors were not allowed, and all patients received Carafate® (saucifate). Third, enteral feeding was commenced in all our patients at the start of the study, whereas the duration of prior enteral feeding in their study was variable, with one patient receiving prior enteral feeding for up to 188 days. Fourth, they only obtained one gastric pH and culture while the patient was on continuous enteral feeding for comparison to subsequent samples obtained while intermittent feeding was administered. Thus, their data on continuous feeding may have been subject to sampling error, since they only had one sample per patient. Finally, they included Candida spp as a significant organism, despite the fact that Candida very rarely causes pneumonia.

A potential role for intermittent enteral feeding was supported by data reported by Lee and Jacobs.2 They reported a lower incidence of nosocomial pneumonia in patients receiving intermittent enteral feeding compared to continuous enteral feeding; however, they used a historical control group and did not report data on gastric colonization.

The disparate results by Spilker and colleagues,1 Lee and Jacobs,3 and us4 suggests that the final chapter on the role of enteral feeding in gastric colonization has yet to be written. Further studies are needed to assess the importance of gastric colonization in the pathogenesis of ventilator-associated pneumonia and specifically to assess the utility of intermittent enteral feeding schedules in decreasing gastric colonization, and ultimately nosocomial pneumonia.

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REFERENCES

Another Important Detail of Percutaneous Dilatational Tracheostomy Technique

To the Editor:

We enjoyed reading the excellent article of Marx and colleagues (September 1996)5 about some of the details in the technique of percutaneous dilatational tracheostomy. However, we feel that another important detail is lacking. Because changing the cannula may be a potential source of complications, the authors have stated that before 2 weeks, a stoma tract has not developed sufficiently to allow safe recannulation.1 However, they may not have prevented unscheduled changes. In our practice, we routinely change tracheostomy tubes safely by 1 week after percutaneous tracheostomy. In some instances, we have performed unplanned changes of cannulae because of problems with the endotracheal cuff, even a few hours or days after percutaneous tracheostomy. In all cases, we insert the new tracheostomy tube using the same model of sterilized guidewire, guiding catheter, and blue dilator. The sterile package accompanies the patient until his or her tracheostoma has a secure, mature tract. We suggest that a simpler (and cheaper) variety of these sets containing only a guidewire with “J” tip, a guiding catheter, and a single blue dilator (18F, 21F, 24F, or 28F) for changing tracheostomy cannulae should be commercially available. We think changes of tracheostomy tubes at any time would be safer using this method. We hope this simple procedure can be added to the accurate observations of the authors in order to improve the excellent results of this technique.

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To the Editor:

On occasion, we have had to reinsert the tracheostomy tube shortly after dilatation. We have utilized the introducer set in the fashion that you describe.

It is quite easy to access the stoma and redilate the tract. It is not usually necessary to use all of the dilators, and as you suggest the tracheostomy tube can be reinserted without difficulty.

In all cases we have temporarily secured the airway with orotracheal intubation. This prevents the loss of airway should some difficulty be encountered with reinsertion of the tracheostomy tube. The patient is therefore not placed at any risk.

We agree that a new kit could be manufactured, but we are certainly not sure if this is feasible for the company.

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CHEST / 111 / 4 / APRIL, 1997 1475