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To the Editor:

We appreciate the comments of Dr. Teramoto and colleagues on our article in CHEST (February 2002).1 We agree that abnormal swallowing physiology and dysphagia are common in patients with COPD.2,3 In fact, we have reported that dysphagia is present in 17 to 20% of patients with moderate and severe COPD.4,5 Our objective was to demonstrate that patients with stable COPD and hyperinflation are at increased risk of aspiration. Although patients with COPD demonstrated a lower resting laryngeal position, none of the 20 patients evaluated by a very comprehensive assessment of swallowing physiology had evidence of laryngeal penetration and aspiration. To our surprise, 45% of patients exhibited frequent swallowing maneuvers that seemed protective in nature. Whether these maneuvers are protective in acute exacerbation of COPD, where patients may develop worsening of hyperinflation and tachypnea, remains to be elucidated. In a small study by Shaker et al.,6 10 patients were evaluated by a videofluoroscopy swallowing study both during acute exacerbation of COPD and after recovery from exacerbation. The investigators demonstrated that during exacerbation the patients swallowed significantly more in the inspiratory phase and resumed their respiration more with inspiration. However, none of the patients demonstrated any evidence of laryngeal penetration and gross aspiration.

The new methods for detection of swallowing disorders described by Teramoto and colleagues seem useful for a rapid bedside evaluation technique to assess a patient’s risk of aspiration.6 However, this technique cannot identify the exact location and mechanism of swallowing dysfunction; therefore, it should not replace a comprehensive videofluoroscopic swallow evaluation in research protocols.

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Complete Interatrial Block Revisited

To the Editor:

Although it has been many years since the article by Drs. Wanchum and Zhoori2 appeared in CHEST, I feel one of their conclusions—ie, that the narrow P waves (NPs) such as P5 and P6 of strip 2B; P2, P3, and P10 of strip 2C (Fig 1); and P5 and P10 of strip V1 (Fig 2) are due to the fact that during sinus rhythm (SR) the left atrium did not depolarize—is incorrect and should be challenged for two reasons. Both reasons show that the NP focus is not the sinoatrial node (SAN), as the authors contend, but rather a different focus.

The first reason is that the rate of the NPs, which is nearly constant, is definitely faster than the rate of the wide P waves (WPs) [ie, those SAN P waves with a block or delay in Bachmann bundle (BB)], as can be seen from the fact that the P5–P6 interval of strip 2B and the P2–P3 interval strip of 2C is less than the P5–P7 or P7–P9 intervals of strip 2C.

The second reason is that the NPs occur only after a retrograde P wave fails to appear immediately after a functional beat and not with an intermittent randomness as implied by the authors. This indicates that the NP focus lacks entrance block protection from the retrograde P waves, whereas the SAN has entrance block protection. The latter can be seen by the fact that, in strip 2C, the length of the WP SAN cycles ended by P7 and P9 are the same length, despite the fact that the times from their immediately preceding retrograde P waves are different.

As the NP focus is not the SAN, the possibility is also ruled out that the NPs are the result of SR with a complete recovery in BB(2) with almost simultaneous activation of both atria.

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