On “Who Will Teach the Medical Students”

To the Editor:

I wish to compliment Dr. Block on his editorial, “Who Will Teach the Medical Students” (CHEST 1995; 107:1). I cannot recall a more exact, concise, and didactic presentation of the deterioration of undergraduate medical teaching. Possibly, the devastating effects are not as visible as they should be due to the excellence of many resident and fellowship programs. In Brazil we have few Fellows but many people with master of science and doctorate degrees in clinical medicine. They play the approximate role of an American Fellow.

In Brazil there is no hope of adjusting university teaching staff (medical or otherwise) salaries to reality. Therefore, we have to make do with what we have.

We are in the process of trying a tutorial system for the undergraduate teaching of pulmonology. In reality, it is based on the apprenticeship experience. Our 50 students will be divided into 10 groups. Each group will spend 6 weeks with selected role models: production line clinicians (there are no others), young, enthusiastic, successful, and with at least a Master of Science (MSc) degree, which requires original research abilities and a University-approved running project. The students will really follow their instructor in whatever he is doing. The instructors will be thoroughly trained about the core objectives, problem solving abilities, and behavioral attitudes the students are expected to achieve. A few selected topics will be reviewed in lecture form. We thus hope to combine the old apprenticeship with the research-based approach to problem solving, which we believe is one of the most important features of the post-Flexnerian era. We would strongly welcome interchange with institutions with a similar program since, to our knowledge, none has been published.

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Bronchodilating Effects of Combined Therapy With Clinical Dosages of Ipratropium Bromide and Salbutamol for Stable COPD

To the Editor:

I noted with interest the article by Ikeda and colleagues (CHEST 1995; 107:401-05) who demonstrated the additive bronchodilating effects of inhaled β-agonistic and antimuscarinic agents in patients with chronic airway obstruction. Apparently, the antimuscarinic doses were on the plateau of the dose-effect curves since the two doses that were given produced the same bronchodilator response. It is more difficult to be certain that the β-agonists also produced equivalent and maximal effect given the design of the study. However, given my experience with these agents, they might well have been equivalent. If they were, the authors might well have demonstrated additive effect on a serial basis, ie, the two agents may have been acting predominantly at different levels of the tracheobronchial tree.

Many years ago, I and my colleagues provided evidence in normal subjects that an antimuscarinic agent (atropine by inhalation in those days) and a β-agonist (isoproterenol by inhalation) acted on different sites within the airways.1,2 Antimuscarinics appeared to have a predominant effect on more central airways, and β-agonists appeared to have greater peripheral effects. When given in combination, there appeared to be a serial additive effect. Using maximal expiratory flow (Vmax) rates, we demonstrated that the β-agonist produced the same degree of bronchodilatation while breathing air but with different effects on Vmax while breathing 80% helium–20% oxygen (heliox). With the lungs filled with heliox there was a significantly greater increase in Vmax following a β-agonist than after atropine, ie, isovolumic density dependence of Vmax was high after β-agonists and low after antimuscarinics. When the two agents were given together at maximally effective doses, Vmax was greater than with either agent alone and density dependence of Vmax became intermediate in magnitude. We interpreted the results as follows: antimuscarinics were having a disproportionate dilating effect on larger upstream airways such that more of the constant driving pressure (static lung recoil pressures were unchanged) was then dissipated across smaller upstream airways where flow is less dependent on gas density. β-Agonists were having a greater dilator effect on smaller upstream airways such that more of the constant driving pressure would now be dissipated across large upstream airways in which flow is more dependent on gas density. We independently reached the same conclusion by analyzing critical alveolar pressures from isovolumic pressure flow curves.1 All of the above was based on the equal pressure point analysis, but now could easily be adapted to the choke point theory and arrive at the same conclusions.

In another study,2 again using normal subjects, we used sub-maximal flow techniques and demonstrated a much larger increase in anatomical dead space after atropine than with isoproterenol, indicating a greater central effect of the antimuscarinic agent. The β-agonist caused a change in closing volume, whereas the antimuscarinic did not, indicating a greater peripheral effect of the β-agonist. Both drugs produced identical overall dilation as assessed by change in specific conductance.

We never extended our ideas to patients because inter- and intra-regional nonhomogeneities in disease would have prevented reliable assessment of localization of responses using the techniques that we used to reach our conclusions in normal subjects.

These ideas, which were not only novel at the time but are still novel and untested in disease, may well have a life in relevance if the two drugs used by Ikeda et al (ipratropium and salbutamol) were each given at maximally effective doses. If so, Ikeda and colleagues may well have done the study that we should have done 20 years ago. Therefore, my question to the authors is whether the higher β-agonist dose produced the same bronchodilator effect as the lower one without the antimuscarinic agent? If so, our previous observations would be pertinent to their interpretations. If not, they still have a chance to test these ideas in a disease state where they (the ideas) would have some conceptual importance in the management of such patients.

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REFERENCES
