Is Anyone Paying Attention?

Work force studies often reflect common prejudices, cost-saving philosophies, rumor and innuendo, but little data. An exception to this rule exists in the recently published Committee on Manpower of Pulmonary and Critical Care Societies (COMPACCs) study.\(^1\) As I read it, there is a shortage of pulmonary and critical care specialists now and this shortage will get worse in the future. These conclusions are based on real survey data with excellent physician participation.

This is no surprise to me, since I have some personal observations to back up these conclusions. In 28 years of training pulmonary fellows, I do not remember more than two who had any difficulty in finding a job. Mostly, the fellows were besieged with job offers. In reviewing the personnel services of this journal for the past 8 years, there are usually between 10 pages and 20 pages of advertisements for positions available as opposed to one or two total positions sought.

In the 1990s, common wisdom told us that we were training too many specialists. Perhaps this was true for many specialties, but it was never validated for the pulmonary and critical care physicians. Medical schools tried to cut the number of fellows trained in all specialties, including ours.

I must apologize for picking on the state of Florida in this discussion, but I live there, was involved in all the matters that occurred, and have little knowledge of affairs in other states. Not only did the medical schools cut the numbers of fellows trained, but the American Lung Association (ALA) suddenly prevented the Florida Lung Association (or any other state associations) from funding clinical fellows. I was distressed by this decision because in 1979, the division heads from the three medical schools in Florida initiated the Pulmonary Wintercourse sponsored by the Florida Thoracic Society and the Florida Lung Association. The single purpose for founding this course was to raise money to fund the training of clinical fellows. This goal was accomplished, and the Florida Lung Association funded the training of one fellow in each of the three institutions for 18 years. Since 1997, however, funding has been restricted by the ALA to research, including money generated by the Wintercourse. I was one of the three division heads that started the course. I felt betrayed in 1997 when the money could not be used for the purpose that we originally intended. I strongly believe that the ALA restrictions should now be lifted in the wake of the publication of recent data that support more fellowship training. That was the original intention, but it was sidetracked by national events. The decision should be reversed. In fact, I believe that clinical fellowship training in general should be funded again by both the National Institutes of Health and the ALA in every state.

Sue Pingleton has written recently of the merits of the COMPACCs study.\(^2\) There will only be merits from this study if behavior changes and the gap between needed specialists and practicing pulmonary and critical care physicians is closed by training. Even if a follow-up study is done, it will report no new information if we do not act now.

With new leadership in the ALA, I ask why the local funds are still restricted? I am told that the Professional Education and Research Committee of the Florida Lung Association cannot allocate all the monies available now since the ALA must approve everything that is funded, even at the local level. This includes all research grants and excludes fellowships. Does this make any sense in 2001? I am sure that this question applies not only to Florida but to the whole country. We can make a difference in the manpower status, but we must do the right thing. Is anyone paying attention?

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REFERENCES
1. Angus DC, Kelly MA, Schwartz RJ et al. Committee on Manpower for Pulmonary, and Critical Care Societies. Current and
**Men Are From Mars, Women Are From Venus**

**Lessons To Be Learned From the Differences Between the Sexes**

John Gray’s famous book, *Men Are From Mars, Women Are From Venus*, focuses on the differences between men and women.1 This book deals with relationships between men and women using a metaphor that Martians (men) and Venusians (women) met, fell in love, and developed loving relationships; on moving to Earth, they forgot they were from different planets. Subsequently, their relationships deteriorated. Maybe the sexes are more different than we think, and maybe our understanding of disease states will benefit from closer examinations of the variations of specific diseases between the sexes.

In the context of Gray’s book, obstructive sleep apnea (OSA) is a Martian’s disease. The reason for this is not completely understood. The study in this issue of CHEST (see page 1442) by Mohensin compares upper-airway size utilizing the acoustic reflectance technique in a group of men and women referred to a university sleep center. Similar studies2–3 utilizing other imaging techniques in normal subjects have shown that although women have smaller pharynges when seated, on recumbency the differences between men and women disappear. The current study population, because they were a group of patients at risk for OSA, were more obese than patients in prior studies (mean body mass index [BMI] > 33 kg/m²). The authors showed that although the women had higher BMIs and smaller pharynges than the men, they had less severe OSA. Additionally, the size of the pharynx (< 3.2 cm²) correlated with the severity of OSA only in men. This article suggests there is something inherently different about the properties of the upper airway in men compared to women.

Collapsibility of the upper airway depends on its size, the surrounding muscle tone, and the characteristics of the tissue. All of these are interrelated: if the muscles that surround and dilate the airway tone are lax, the airway will narrow; if there is an increased amount of fat in surrounding tissues, the airway may be compressed. Other studies have investigated the gender differences related to muscle tone in the upper airway.4,5 Upper-airway resistance increases with sleep onset, and this results in a reduction in minute ventilation. Awake genioglossus electromyogram (EMG) activity in women may be related to hormonal status (luteal vs follicular phase of the menstrual cycle vs postmenopausal state), difference does not seem to affect upper-airway resistance.4 There appears to be no significant difference in muscle tone in upper-airway dilators (genioglossus and tensor veli palatini) between men and women during sleep, although men have a higher upper-airway resistance.5 Unfortunately, these studies were done in normal subjects, so how this extrapolates to OSA patients is unclear.

Sex hormones have also been thought to influence the development of OSA. A recent study by Bixler et al6 showed that OSA was much more prevalent in postmenopausal women who were not receiving hormone replacement therapy (HRT), compared to postmenopausal women receiving HRT or premenopausal women. HRT has also been shown to decrease sleep-disordered breathing indexes (apnea/hypopnea index [AHI]) in postmenopausal female patients.7 Other reports8,9 have shown that OSA is more prevalent in women with androgen excess, for instance, polycystic ovary syndrome. New onset of OSA developed in a woman who received exogenous testosterone.9 This suggests testosterone may also play an important role.

Other investigators have examined the surrounding tissue in the upper airway. MRIs of the upper airway have shown differences between the sexes; nonobese male subjects were shown to have more fat in their necks compared to the rest of their bodies.10 Another study,11 looking specifically at physical examination findings, showed that male patients with a narrowing of the lateral pharyngeal walls and/or tonsil enlargement were at greater risk for OSA, but no specific physical examination finding was predictive of OSA in female subjects.

Polysomnography findings have also been shown to differ between men and women with OSA. Not only do women have lower AHI results than men, most of the difference in AHI between men and women occurs during nonrapid eye movement (NREM) sleep. The AHI in patients during rapid eye movement (REM) sleep is equal in men compared to women, suggesting that whatever protects women from upper airway collapse in NREM sleep disappears on entering REM sleep.12,13

Is there a common theme to explain the gender difference in OSA? The size of the airway seems to make a difference in men, and this may be an effect of a difference in muscle tone (lower in men) and tissue characteristics (floppier in men). The difference in tone may be abolished when REM sleep is

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1 Pingleton SK. Committee on Manpower of Pulmonary, and Critical Care Societies: a report to membership. Chest 2001; 120:327–328

2 Mohensin S. Differences Between the Sexes: Lessons To Be Learned From the Men Are From Mars, Women Are From Venus. CHEST 2017; 151:437–442

3 Pingleton SK. Differences Between the Sexes: Lessons To Be Learned From the Men Are From Mars, Women Are From Venus. CHEST 2017; 151:437–442

4 Bixler EO. Sex hormones and sleep-disordered breathing. J Clin Endocrinol Metab 1999; 84:1–2

5 Bixler EO. Sex hormones and sleep-disordered breathing. J Clin Endocrinol Metab 1999; 84:1–2

6 Bixler EO. Sex hormones and sleep-disordered breathing. J Clin Endocrinol Metab 1999; 84:1–2

7 Bixler EO. Sex hormones and sleep-disordered breathing. J Clin Endocrinol Metab 1999; 84:1–2

8 Bixler EO. Sex hormones and sleep-disordered breathing. J Clin Endocrinol Metab 1999; 84:1–2

9 Bixler EO. Sex hormones and sleep-disordered breathing. J Clin Endocrinol Metab 1999; 84:1–2

10 Bixler EO. Sex hormones and sleep-disordered breathing. J Clin Endocrinol Metab 1999; 84:1–2

11 Bixler EO. Sex hormones and sleep-disordered breathing. J Clin Endocrinol Metab 1999; 84:1–2

12 Bixler EO. Sex hormones and sleep-disordered breathing. J Clin Endocrinol Metab 1999; 84:1–2

13 Bixler EO. Sex hormones and sleep-disordered breathing. J Clin Endocrinol Metab 1999; 84:1–2