A Different View of Academic Medicine

Money is driving everything! Learned professors have written recently of downsizing academic clinical operations and of establishing academic managed-care networks. These responses are intended to make the academic health center more competitive with the burgeoning health maintenance organizations and private sector managed-care health delivery systems. Many medical schools have established branch operations with neighborhood clinics and “feeder” clinics to continue referrals to the tertiary medical centers. Direct competition with the private sector has resulted in animosity on the part of the private physicians. All of this activity results from the need to survive financially in the medical field of the 1990s.

Is this trend an inevitable consequence of health-care reform in the United States? Will the academic health centers be forced to compete with the managed-care networks already in existence? Can the academic health centers survive such a competitive venture? I submit that the answer to all of these questions should be no! The basic reasons for the existence of academic health centers is very different from that of the managed-care sector of medicine and includes many more missions than just seeing patients and making money.

Medical students must be educated and trained. Both basic and clinical research must be performed. The advances derived from this research must be adapted to improve patient care. In fact, I have always viewed the purpose of any department or division in an academic health center as being complementary to and not competitive with the private sector. If the academic unit is doing its job properly, even physicians trained as little as 2 years earlier will be several years out-of-date. The advances made in the academic center should assure continual referral of patients whose care will be improved by the advances resultant from basic and clinical research. Examples of this training process are numerous and include transplantation of various organs, immunologic treatments for type 1 diabetes mellitus, intrabronchial stent placement, etc.

The evidence that specialty care in academic health centers is more advanced than in the private sector is easy to come by; but the reimbursements that should result for such care are hard to acquire. Insurance companies take forever to assign a reimbursement for a new procedure they have not hitherto encountered. Minor but important variations on established procedures, i.e., transbronchial needle aspirations, cannot be billed as additional procedures. In a recent editorial in the New England Journal of Medicine, the editor stated, “Insurance companies should not be allowed to reap the benefits of the academic centers without paying the price”.

Whether the price will mean a higher reimbursement for each individual component of care or a premium tax on the insurance companies is immaterial to me. We must recognize that treating academic centers the same as private sector managed-care will impede the advances in medicine for which the United States is known. There is evidence that up-to-date cutting edge care is superior. Should not the reimbursement be superior also and sufficient to allow the academic specialist to augment but not compete with the primary care physician?

My view of academic medicine is old-fashioned, I admit. The most difficult, complicated, perhaps hopeless cases should be sent to the academic medical center. Teaching services should exist where educated academic physicians can teach medical students and house staff the principles of modern medicine and in so doing treat and cure these complicated patients. Follow-up care is best provided by the private sector. Lastly, the support for such academic ventures should be provided by relief from bureaucratic constrictions that make no sense, proper reimbursement for care that is out of the ordinary, and the financial recognition that teaching and research are worthwhile endeavors and are the vehicles that have driven American medicine to the forefront.

Although this argument may seem idealistic or old-fashioned to many people in the 1990s, that doesn’t make it wrong. To effect such a system would require the cooperation and understanding of multiple institutions. Certainly the sponsors of the academic health center (private, state) must recognize the importance of the venture and financially support it. Second, the insurance companies must ante up in some way. Third, the federal government should not forget that training physicians costs money and should continue the subsidy for training that has previously existed. I would, however, direct the payments to the medical school rather than the hospital administration as has been done in the past. Finally, the country should...
acknowledge that the academic health system has produced the finest medicine in the world. It is not the cheapest, but it is the best. Without an understanding of what has produced this lofty position, we will degenerate into a pure competition with private practice and the new advances will cease—a process that I believe is already happening.

A. Jay Block, MD, FCCP
Gainesville, Florida

REFERENCES

1 Iglehart JK. Rapid changes for academic medical centers. New Engl J Med 1994; 331:1391-95
7 Block AJ. A problem in academic internal medicine: we have taken the teaching out of the teaching hospital. Pharos 1992; 55:28-30

What We Don’t Understand About Ozone Effects

Ozone (O₃), a highly reactive triatomic form of oxygen (O₂), is inhaled as the predominant component of photochemical air pollution. Ozone is categorized as a secondary air pollutant, being derived from photochemical reactions of nitrogen oxides and volatile organic compounds in the troposphere. Although significant progress in reducing O₃ levels has occurred, even in Los Angeles, millions of Americans continue to be exposed to ambient concentrations above the health-based federal standard, which is 0.12 part per million (ppm) for 1 h, not to be exceeded more than once a year. These “exceedances” may occur at least a few days per year in numerous urban and rural areas throughout the country, and more than 100 days annually near Los Angeles. It is not surprising that O₃ has generated a great deal of public health concern, continuing regulatory efforts, and research involving animal toxicology, controlled (clinical) exposures, and epidemiology.¹⁻³

Human volunteers exposed to controlled O₃ levels ≤0.40 ppm experience short-term decrements of lung function that vary widely between different subjects but are reproducible over time in a given individual.⁴ McDonnell et al⁵ retrospectively investigated numerous characteristics that might predict individual differences in acute response (FEV₁) among 290 O₃-exposed healthy male volunteers aged 18 to 32. Besides O₃ concentration, only age was a significant predictor, ie, response decreased with increasing age. The carefully performed, prospective study by Aris and coworkers (see page 621) confirms and extends previous work regarding the poor predictability of individual responses to O₃. Methacholine responsiveness did not predict O₃-induced lung function responses, nor did respiratory symptoms correlate well with the lung function changes due to O₃.

The above findings indicate that we still have much to understand about O₃-related effects. Responses to O₃ may be measured in terms of decreases in FEV₁ or increases in symptoms, airway resistance, “nonspecific” airway reactivity, airway inflammation, lung permeability, or mucociliary clearance. However, many of these responses do not appear to be related when concurrently measured;¹⁻⁷ this, in part, may be related to differences in response kinetics to O₃. Even when portions of a neurophysiologic response mechanism are understood, the initiating events remain unknown. For example, inhaled O₃ might stimulate airway C-fibers by a direct irritant effect, or indirectly by releasing mediators, or both. Multiple endogenous and exogenous factors influence the biologic response to O₃, including genetics, dosimetry (the quantity of O₃ available to react at a given location in the respiratory tract), nutrition, preexisting lung disease, and recent history of exposure, eg, to O₃, other pollutants, or smoking. Rapid shallow breathing is a typical physiologic response to O₃ exposure, which may protect against pathologic effects by altering dosimetry to reduce the overall dose to lung tissue. Thus, differences in ventilation patterns and resulting dosimetry might contribute to interindividual variability in O₃ responses.⁸

What does all this mean to physicians, patients, and the general public? For researchers concerned with biomedical or public health issues, questions about O₃ effects now appear more complex, and answers appear more tentative than before. Is there a common mechanism underlying the diverse manifestations of O₃ response? Are inflammatory and other changes in the lungs physiologically adaptive or toxic? Are identifiable subgroups of the population consistently extrasusceptible to O₃ effects? Which acute responses predict chronic O₃ effects, if any? Lacking clear answers to these questions, clinicians