nurse was present at the bedside in about 25% of UE cases,² nursing presence alone does not guarantee prevention.

We conclude that further study to identify risk factors for UE, greater attention to basic preventative measures, and continued emphasis on timely elective extubation should help reduce the incidence of UE. However, on considering the vast number of intubated ICU patients, the determination of some agitated patients to remove the ET tube, and the realities of ICU patient surveillance, we anticipate that the problem will be with us for years to come.

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REFERENCES

Chest X-ray Screening for Lung Cancer

To the Editor:

Strauss and colleagues (CHEST 1995;[suppl]107:270S-79S) claim that periodic chest x-ray screening for lung cancer should be reconsidered because two randomized trials (their references 10-13) have shown improvements in stage distribution, resection rate, and survival. With convoluted reasoning, they dismiss overdiagnosis and lead-time bias as explanations for these results. They discard the well-accepted dictum that screening has had no impact on lung cancer population mortality rates, which are conclusions supported by the two studies they reviewed.

Strauss et al published a similar article in 1993 in CHEST,¹ which was branded by Parkin and Pisani of the International Agency for Research on Cancer² as mere “opinions, unsubstantiated by any data.” Strauss et al offered no rebuttal to the criticisms, criticisms with which I completely agree.

Strauss et al make much of the fact that the screened groups had a higher incidence of lung cancer not explained by lead-time bias. True, but then they reason that lead-time bias predicts only an initial excess of cases due to length bias! They are confused. Length bias only applies to the prevalence cases picked up on the first screening when the more slowly growing cancers are overrepresented. Both studies eliminated the prevalence cases. Lead-time bias has nothing to do with length bias and persists throughout the screening period.

They make the assumption that all lung cancers are life-threatening if not effectively treated. The fact is that malignant changes in the bronchial mucosa tend to be multifocal. This was established in a classical study by Auerbach et al³ (not mentioned by Strauss et al) and provides a reason for some overdiagnosis by screening, especially among cases detected only by sputum cytology. How often was an indolent focus of carcinoma in situ resected that was not destined to cause death, especially in the face of competing causes of death? And what about underdiagnosis in the control group?

Some of their arguments are specious and the rest are speculative. A thorough discussion of all the deficiencies in their article would take more space than a letter is allowed.

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REFERENCES

To the Editor:

Our two articles (CHEST 1993,[suppl]103:337S-41S; and 1995; [suppl]107:270S-79S) on lung cancer screening have stimulated three Communications to the Editor. Each is highly critical of our position that existing randomized trials support clinically important benefits from periodic chest x-ray (CXR) screening. However, each is particularly critical of our central argument that CXR screening does not lead to the significant overdiagnosis of lung cancer.

Parkin and Pisani (CHEST 1994; 106:977) assert that our arguments challenging the overdiagnosis hypothesis represent “only opinions, unsubstantiated by any data.” While we acknowledge that statistically “overdiagnosis in a screened group can plausibly account for . . . ‘missing cases’ in a control group” (CHEST 1995;[suppl]107:270S-79S), we present three lines of evidence that we believe raise substantial doubts about the tenability of the overdiagnosis hypothesis in lung cancer. First, we show that overdiagnosis is not biologically plausible in the context of the epidemiology and known clinical virulence of lung cancer. Second, we show that there are very few long-term survivors among screen-detected lung cancers who do not undergo optimal therapy.

Third, we present autopsy evidence. While in prostate cancer, two thirds of men over the age of 60 have “latent” prostate cancer discovered at autopsy, a comparable phenomenon does not appear to exist for lung cancer. We cite the Yale experience, which reported only a 0.5% prevalence of “surprise” lung cancers among 3,286 autopsies.¹ Moreover, most of these surprise cases were not latent cancers, because 58% had regional metastases or distant metastases or both, and 37% were too ill for medical evaluation, suggesting that some individuals discovered to have surprise lung cancer at autopsy had died of lung cancer.

Parkin and Pisani (CHEST 1994; 106:977) believe that “overdiagnosis is a very plausible explanation for the excess cases in screened populations.” To support their position, they cite an au-

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