Chest X-ray Screening Does Not Improve Outcome In Lung Cancer

To the Editor:

Strauss and colleagues (CHEST 1995; 107:2708-795) continue to “reinterpret” the overwhelmingly negative evidence concerning the utility of chest x-ray screening for lung cancer. Since their previous attempt, they have discovered a second randomized trial which makes their thesis that the negative results are due to chance highly improbable, and they have moved on to the equally implausible idea that these negative results are from a failure of randomization.

With rather more careful research, they would have identified five trials comparing populations undergoing intensive screening, with control groups having fewer, or no, chest radiographs done: the Kaiser Permanente study,7 the North London Study,3 the Mayo Lung Project,4 the Czech trial,5 and the Erfurt County Study.6 None showed any mortality reduction in the intensively screened group compared with the controls, and the latter four all showed a higher incidence (more cases) in the screened group compared with the control group. The first four of these studies were based on random allocation to study and control groups, and there was no evidence of differences in important variables, eg, smoking, between them. Confounding (“imbalance in covariates”) in randomized studies is possible but very unlikely to have occurred in the same direction in all four.

Strauss et al also try to support their hypothesis that confounding occurred in two trials—in spite of randomization—by incorrectly quoting the results of the studies. In the Czech trial the incidence difference between the two groups did not triple during the 3-year period that followed the intervention; in fact, 26 cases are the final cumulative difference for the entire study period (therefore including intervention). Seventeen more cases were detected during intervention in the screened group, and only nine during follow-up.3 Similarly, we could not recognize the figures reported by Strauss et al on the Mayo study in the article by Fontana et al.4 During the follow-up period, nine excess cases were detected in the control group rather than the screened one.

As we have already pointed out,7,8 overdagnosis is an entirely feasible and indeed likely explanation for the findings in these studies. In the Mayo Lung Project and the Czech trial, the excess incidence occurred in the screening phase and not—as stated by Strauss et al—during the follow-up period. There is no need to advance overdagnosis to explain favorable tumor size, stage, and survival in screened populations. These are the predictable consequences of lead time and length biased sampling.

Finally, it is disingenuous in the extreme to expect decreases in smoking prevalence, occurring mainly in young generations, to impact immediately on lung cancer deaths, which occur predominantly in the elderly, and it is unfair to present the trends in lung cancer incidence and mortality to hide the declines occurring in men since at least 1988.9 In fact, the trends in lung cancer incidence and mortality in the United States are very well predicted by cohort-specific smoking data.10

There is no need for more consensus conferences to review the evidence relating to chest x-ray screening. At present, this is completely negative. It can only divert attention from the need to pursue a vigorous approach to primary prevention through curbing tobacco use.

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REFERENCES


Late Intracapsular Hemorrhage in an Anticoagulated Patient With a Breast Implant

To the Editor:

Atrial fibrillation is a common disorder with an incidence that increases with age such that 3% of the population over 55 develops the arrhythmia.1 Anticoagulation is now routinely recommended for most patients with atrial fibrillation and a satisfactory hemorrhagic risk.2 Between 1 and 2 million women have had breast implants since their introduction 30 years ago. As the population of women who have received breast implants ages, a significant number can be expected to develop atrial fibrillation and be considered for anticoagulation. We have observed one such patient who developed a late intracapsular hematoma while anticoagulated 15 years after breast reconstruction with a silicone gel prosthesis.

A 52-year-old woman underwent a two-stage reconstruction of her right breast with implantation of a silicone gel prosthesis 10 years after a right simple mastectomy with axillary node dissection for adenocarcinoma of the breast. With the exception of the development of a Baker's class 2 contracture, she did well over the subsequent 15 years until she developed paroxysmal atrial fibrillation. She was anticoagulated and her international normalized ratio (INR) was maintained between 1.5 and 2.7. Approximately 4 months after initiating anticoagulation, she noticed discomfort and a change in the size and shape of her right breast without a history of breast trauma. A diagnosis of intracapsular hemorrhage was made, and she underwent an evacuation of hematoma, removal of a ruptured implant, and insertion of a saline breast prosthesis (McGhan; McGhan Medical Corp; Santa Barbara, Calif). After a recovery period of 10 weeks, anticoagulation was reinitiated and the

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