These findings should stimulate critical care physicians to re-examine at the role of routine dopamine infusion in such patients. Concomitantly, one must reassess the negative views on norepinephrine infusion that have developed. Prior bad experience likely reflected the combination of excessively high doses with inadequate effective plasma volume replacement. This combination led to a restoration of blood pressure but not perfusion in patients who were noted to have “cold, clammy skin” and oliguria that progressed to renal shutdown and death. Indeed, the practice of adding norepinephrine to the ongoing infusion was so feared that this treatment was dubbed a “lethalpheid infusion.” A relook at the role of norepinephrine infusion in a setting where more accurate invasive monitoring allows for frequent assessment of cardiac index, peripheral vascular resistance, oxygen delivery, and central filling pressures is needed. Use of the Frank-Starling curve to identify when high central filling pressures are indicative of lung failure but not heart failure will facilitate these future studies.

A frequent misconception is that the increased urine output observed in response to a low-dose dopamine infusion indicates a dopaminergic renal receptor which facilitates renal vasodilation and, thus, enhanced renal perfusion independent of cardiac index. The authors have demonstrated that this increase in urine output also occurs with norepinephrine infusion. Their data support the conclusion that the increase in urine output in response to a low-dose infusion of both agents results not from a renal receptor but from the traditional baroreceptor responses that shut off the release of antidiuretic hormone from the pituitary. No study in man using traditional methods for measuring renal blood flow has shown an increase in renal blood flow independent of an increase in cardiac output after low-dose dopamine or norepinephrine infusion. Unpublished studies from our laboratory show that renal blood flow is directly related to cardiac index after the addition of dopamine; thus, the increased urine output is explained totally by the antidiuretic hormone effect. The authors must be congratulated on this fine project, which should lead to many similar prospective assessments of these agents.

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Plaques, Cancer, and Confusion

Asbestos research has been fraught with conflicting claims of an association between pleural plaques and lung cancer. Nineteen studies have been published,1-19 and only four studies1,4,5,9 have claimed to show an association between the risk of lung cancer and pleural plaques. A new study by Hillerdal published in this issue of Chest (see page 144), is the 20th study to re-evaluate this problem.

The research hypothesis is simple. Asbestosis is associated with lung cancer, and therefore, some patients with asbestos exposure may have subclinical asbestosis, and an increased risk of lung cancer. Pleural plaques are commonly seen in asbestos-exposed individuals, and therefore, may be associated with an increased risk of lung cancer due to asbestos exposure. A good epidemiologic study must adequately control for cigarette smoking intensity as well as duration, race, sex, fiber type, asbestos exposure, and exposure to other potential carcinogens in the work place. At stake in all these studies is a much larger issue. If patients without asbestosis, who have only pleural plaques as evidence of asbestos exposure, do not have an increased risk of cancer; then this would support the concept that a minimal threshold dose sufficient to cause asbestosis is necessary for the promotion of lung cancer.

Studies such as Hillerdal’s are greatly weakened by the inability to accurately quantitate asbestos exposure. Significant bias in smoking histories may occur due to differences in smoking habits such as smoking to the end of a cigarette, deeper inhalations, and the use of nonfiltered cigarettes, as well as bias from a social class or occupational group.20,21 The problem is further confounded by Hillerdal’s admission that only 10 to 15 percent of pleural plaques found at autopsy are visualized on chest roentgenogram. One-half or more of the radiologically diagnosed plaques will not be confirmed at autopsy. The specificity of the chest x-ray film can be increased by rigid interpretation criteria to be as high as 85 percent using large films,22 but the specificity of using small films is unknown.

Autopsy studies have demonstrated no increase in asbestos body counts, as compared to a control population, in up to 50 percent of patients with pleural plaques.23 Other autopsy studies have found up to 20 percent of plaque cases without any identifiable asbestos.23,24 A carefully performed radiologic survey in a hospital setting found no evidence of asbestos exposure in 19 percent of patients with radiologically identifiable pleural plaques.25 In carefully performed autopsy studies, pleural plaques do not predict asbestosis.24,26 There is only one published study that has adequately matched control subjects for exposure and smoking history in a single-nested case control study.15 This study by Harber et al15 found no increased risk for lung cancer but did not address the question of increased risk of shipyard workers compared with a nonasbestos-exposed population. A recent excellent review by Weiss27 published in Chest found no evidence of an increased risk of lung cancer associated with pleural plaques. This review is recommended...
reading for students of this issue. The study of Hillerdal does not demonstrate a significant enough increase in risk to overcome the experimental limitations in his study. This study is just another study that has failed to demonstrate convincing evidence of a significant risk of lung cancer associated with pleural plaques without asbestosis.

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