3. Milne ENC, Pistolesi M. Quantification of pulmonary blood volume, flow and pressure: principles. In: Reading the chest radiograph: a physiologic approach. St Louis, Mo: Mosby, 1993; 164–202.
4. Milne ENC, Pistolesi M. Quantification of pulmonary blood volume, flow and pressure: practice. In: Reading the chest radiograph: a physiologic approach. St Louis, Mo: Mosby, 1993; 202–241.
5. Short DS. Postmortem pulmonary angiography with special reference to the study of pulmonary hypertension. J Fac Radiol 1956;8: 118–128.
6. Turner-Warwick M. Precapillary systemic-pulmonary anastomoses. Thorax 1963;18: 225–237.
7. Zisman DA, Karlamangla AS, Ross DJ, et al. High-resolution chest CT findings do not predict the presence of pulmonary hypertension in advanced idiopathic pulmonary fibrosis. Chest 2007;132(3):773–779.
8. Devaraj A, Wells AU, Meister MG, Corte TJ, Hansell DM. The effect of diffuse pulmonary fibrosis on the reliability of CT signs of pulmonary hypertension. Radiology 2008;249(3): 1042–1049.

Response

From
Esam H. Alhamad, MD,* and Ahmad A. Al-Boukai, MD†
Pulmonary Division, Department of Medicine (38),* and
Department of Radiology and Medical Imaging,† King Khalid University Hospital, College of Medicine, King Saud University, PO Box 2925 Riyadh 11461, Saudi Arabia
e-mail: *esamalhamad@yahoo.com*

We thank Dr Milne for his thoughtful comments about our recent article (1). The diagnostic utility of small vessel measurements obtained with CT has been demonstrated previously. For example, the ratio between the diameters of the segmental artery and lobar bronchiole in patients with pulmonary hypertension (PH) and chronic thromboembolic disease was found to be elevated in 50% of patients compared with 7% of healthy control subjects (2). In another study on patients with various parenchymal lung diseases, analysis of a combination of main pulmonary artery diameter and the segmental artery-to-bronchus ratio afforded a sensitivity of 86% in terms of detecting PH (3). This sensitivity is remarkably similar to that in our study, in which only pulmonary artery diameter was considered. It is important to note that, in patients with lung fibrosis, traction bronchiectasis (which is common) will likely confound small vessel measurements (4). Further, the clinical significance of tortuous peripheral arteries, which reflect plexogenic arteriopathy and are occasionally detected with CT, is difficult to interpret when pulmonary fibrosis is present. Thus, we believe that neither measuring small vessels nor searching for tortuous peripheral arteries substantially improves sensitivity or specificity of PH detection in the context of fibrotic lung disease.

In his correspondence, Dr Milne correctly describes the roles played by hypoxia-driven vasoconstriction, and structural changes in the small muscular arteries and arterioles, in the genesis of secondary PH. However,as pointed out in our study, exercise-induced hypoxemia caused blood viscosity to rise, and the consequent increase in shear stress affords a potential explanation for progressive dilatation of the pulmonary artery. Such dilatation was thus independent of pulmonary hemodynamic changes noted in the study by Boerrigter et al (5).

PH is prognostically important in patients with ILD. However, the fact that only weak-to-moderate correlation was found between Spo_2 and mean PAP in both our study and the work of others (6) indicates that pulse oximetry cannot be reliably used to screen for PH. Thus, right heart catheterization remains the gold standard for PH diagnosis. Improved noninvasive methods for accurately detecting PH are required.

Disclosures of Potential Conflicts of Interest: E.H.A. No potential conflicts of interest to disclose. **A.A.A.** No potential conflicts of interest to disclose.

References

1. Alhamad EH, Al-Boukai AA, Al-Kassimi FA, et al. Prediction of pulmonary hypertension in patients with or without interstitial lung disease: reliability of CT findings. Radiology 2011;260(3):875–883.
2. Falaschi F, Palla A, Formichi B, et al. CT evaluation of chronic thromboembolic pulmonary hypertension. J Comput Assist Tomogr 1992; 16(6):897–903.
3. Pérez-Enguix D, Morales P, Tomás JM, Vera F, Lloret RM. Computed tomographic screening of pulmonary arterial hypertension in candidates for lung transplantation. Transplant Proc 2007;39(7):2405–2408.
4. Tan RT, Kuzo R, Goodman LR, Siegel R, Haasler GB, Presberg KW. Utility of CT scan evaluation for predicting pulmonary hypertension in patients with parenchymal lung disease. Medical College of Wisconsin Lung Transplant Group. Chest 1998;113(5): 1250–1256.
5. Boerrigter B, Mauritz GJ, Marcus JT, et al. Progressive dilatation of the main pulmonary artery is a characteristic of pulmonary arterial hypertension and is not related to changes in pressure. Chest 2010;138(6): 1395–1401.
6. Zisman DA, Karlamangla AS, Ross DJ, et al. High-resolution chest CT findings do not predict the presence of pulmonary hypertension in advanced idiopathic pulmonary fibrosis. Chest 2007;132(3):773–779.

The Older, the Better

From
Eric N. C. Milne, MD
Green Lake House, Moo Ban Sammakon, 130/88, Soi 1, Moo 1, Trumbol Lumpakkood, Ampuh Thanyaburi, Pathum Thani, 12110 Thailand
e-mail: *encm@hotmail.com*

Editor:

"If you don't have long to live you don't need to worry about the effects of radiation exposure." This seems to be the message in the recent article by Brenner and colleagues (1) in the October 2011 issue of *Radiology*. This would appear to be self-evident and hardly require extensive research to confirm it, but in fact it is apparently not. During a recent session on "Practical Recommendations for Patients Undergoing Chest CT" (2), my colleagues and I made the observation that in more than 100 articles recently reviewed by us on the topic of reducing radiation dose, not one mentioned including the patient's age when consider-

ing the risk of performing thoracic computed tomography (CT). The simplest way to reduce risk is not to perform thoracic CT in younger female patients, *viz,* those who are still menstruating.

In male and older female patients, say (with apologies) those aged 50 years, the risk of breast cancer induction from thoracic CT is so low that the benefit greatly exceeds the cost. In considering radiation risk, therefore, it is essential to look separately at young versus old, at male versus female patients, and, in particular, at pediatric patients.

These groups have widely differing risks and should not be mixed. For example, as Brenner et al have previously pointed out (3), the cancer induction risk of carrying out CT of the chest in a 15-year-old girl is high, whereas that in a 70-year-old man is very low.

One hopes that the authors' continued reinforcement of the differing risks at different ages will lead to their widespread use as a simple and logical way of reducing radiation dose: specifically to those with the highest risk.

Disclosures of Potential Conflicts of Interest: No potential conflicts of interest to disclose.

References

1. Brenner DJ, Shuryak I, Einstein AJ. Impact of reduced patient life expectancy on potential cancer risks from radiologic imaging. Radiology 2011;261(1):193–198.
2. Milne ENC. Practical recommendations for patients undergoing chest CT. Presented at the 42nd Annual Meeting of the Fleischner Society, Heidelberg, Germany, June 23–25, 2011.
3. Brenner D, Elliston C, Hall E, Berdon W. Estimated risks of radiation-induced fatal cancer from pediatric CT. AJR Am J Roentgenol 2001;176(2):289–296.

Response

From
David J. Brenner, PhD,*
Igor Shuryak, MD,*
and Andrew J. Einstein, MD†
Center for Radiological Research* and Cardiology Division, Department of Medicine,† Columbia University Medical Center, 630 W 168th St, New York, NY 10032
e-mail: *djb3@columbia.edu*

We thank Dr Milne for his interesting comments. In fact, the traditional view that radiation-induced cancer risks decrease monotonically with increasing age at exposure (1) has been increasingly challenged in recent years (2–4). This is primarily because one of the main pathways to radiation-induced cancer is now thought to be promotion of preexisting premalignant cells by radiation to a malignant state, and our burden of such premalignant cells increases with age.

Most radiation-induced cancers appear in the "cancer-prone" ages (typically 50–80 years of age), independent of age at exposure (5), so the latency period between radiation exposure and the potential appearance of a cancer decreases markedly with increasing age at exposure. Taking these effects into account, more recent analyses of cancer incidence among atomic bomb survivors suggest that the lifetime risk of radiation-induced cancer is not so different, for example, for exposure at age 5 years versus exposure at age 55 years (3,6). Thus, it is not necessarily the case that "the older the better," as Dr Milne asserts.

In summary, we agree that individual demographics such as age, and also potentially reduced life expectancy (the topic of the article [7] on which Dr Milne comments), should be considered in assessing individual radiation risks. However, radiation-induced cancer risks probably vary less with age at exposure than had previously been thought, which is of particular importance for those patients who are most likely to undergo CT—that is, patients aged 45–65 years (8).

We reiterate that when CT is clinically justified, the comparatively small radiation risk will almost always be outweighed by the potential clinical benefit (9). It is nevertheless prudent to minimize the potential risks from any radiologic exposure, and the considerations briefly described herein suggest that it may not be wise to focus potential risk reduction strategies predominantly on pediatric patients, as Dr Milne suggests. Patients of all ages are important here, including older patients.

Disclosures of Potential Conflicts of Interest: D.J.B. No potential conflicts of interest to disclose. **I.S.** No potential conflicts of interest to disclose. **A.J.E.** Financial activities related to the present article: none to disclose. Financial activities not related to the present article: institution received a grant or has a grant pending from the National Institutes of Health (R01 HL109711-01), GE Healthcare, and Spectrum Dynamics; receives payment for lectures including service on speakers bureaus from Spectrum Dynamics. Other relationships: none to disclose.

References

1. National Research Council. Health risks from exposure to low levels of ionizing radiation: BEIR VII. Washington, DC: National Academies Press, 2006.
2. Heidenreich WF, Cullings HM, Funamoto S, Paretzke HG. Promoting action of radiation in the atomic bomb survivor carcinogenesis data? Radiat Res 2007;168(6):750–756.
3. Preston DL, Ron E, Tokuoka S, et al. Solid cancer incidence in atomic bomb survivors: 1958–1998. Radiat Res 2007;168(1):1–64.
4. Shuryak I, Ullrich RL, Sachs RK, Brenner DJ. The balance between initiation and promotion in radiation-induced murine carcinogenesis. Radiat Res 2010;174(3):357–366.
5. Shimizu Y, Kato H, Schull WJ. Risk of cancer among atomic bomb survivors. J Radiat Res (Tokyo) 1991;32(Suppl 2):54–63.
6. Shuryak I, Sachs RK, Brenner DJ. Cancer risks after radiation exposure in middle age. J Natl Cancer Inst 2010;102(21):1628–1636.
7. Brenner DJ, Shuryak I, Einstein AJ. Impact of reduced patient life expectancy on potential cancer risks from radiologic imaging. Radiology 2011;261(1):193–198.
8. Berrington de González A, Mahesh M, Kim KP, et al. Projected cancer risks from computed tomographic scans performed in the United States in 2007. Arch Intern Med 2009;169(22):2071–2077.
9. Brenner DJ. Slowing the increase in the population dose resulting from CT scans. Radiat Res 2010;174(6):809–815.

Swedish Two-County Trial: Total Mortality Data Are Needed

From
Juan Gérvas, MD, PhD
Equipo CESCA, Travesía de la Playa 3, Buitrago de Lozoya, Madrid 28730, Spain
e-mail: *jgervasc@meditex.es*