generates highly reactive free radicals and is carcinogenic, tera-
genotic, and mutagenic. It has been associated with cancers of the thyroid, bone, lung, breast, and leukocytes. Like lead and asbestos, radiation has no safe threshold. Therefore, performing HRCT scans of the thorax in only those patients who have symptoms suggestive of active PTB seems to be a more pragmatic approach.

The authors have relied heavily on the results of interferon-γ (INF-γ) release assay in a few patients for the diagnosis of active PTB, even in the absence of clinical symptoms. It is well known that higher production of INF-γ correlates to some extent with the activity of Mycobacterium tuberculosis infection, but its low sensitivity and specificity for distinguishing active vs latent TB has been demonstrated by various studies worldwide, and therefore it is not recommended as a diagnostic tool for active TB. We ought to be more judicious with the use of antituberculous therapy, especially when the threat of multidrug resistance and extensively drug-resistant TB is hovering over us.

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Financial/nonfinancial disclosures: The authors have reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

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DOI: 10.1378/chest.10-1249

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Should Patients With Connective Tissue Disease Undergo Exercise Doppler Echocardiography?

To the Editor:

We read with the great interest the recently published article by Kovacs et al1 (August 2010) indicating that exercise Doppler echocardiography (EDE) can be a useful, noninvasive screening tool to detect an increase in systolic pulmonary artery pressure (PAP) during exercise in patients with connective tissue disease. However, in our opinion, there are some interesting issues to be discussed.

According to the recent European Society of Cardiology guidelines on pulmonary hypertension (PH), the definition of PH on exercise is not supported by published data. Thus, no definition of exercise-induced PH can be provided currently. Moreover, US guidelines are consistent with this statement. However, recently published data indicate that more attention should be paid to an exaggerated increase of systolic PAP during exercise, which is suggested to be a marker of early pulmonary vasculopathy in connective tissue disease. Kovacs et al2 revealed that EDE showed a systolic PAP >40 mm Hg during exercise in 26 of 52 patients with connective tissue disease. In our unpublished observations, an increase in systolic PAP of >20 mm Hg at EDE was recorded in 11 (17%) of 65 patients with systemic sclerosis.

In all of the 11 patients with increased systolic PAP, subsequent right-sided heart catheterization (RHC) confirmed systolic PAP increase during exercise. In our opinion, EDE is a potentially useful test to detect “exercise-induced PH” that may represent an intermediate stage between a physiologic response and manifest PH.

Importantly, an elevated systolic PAP on exercise can be caused not only by pulmonary vasculopathy but also by a significant increase in pulmonary venous pressure. Among patients reported by Kovacs et al,1 RHC revealed an elevation of pulmonary arterial wedge pressure >20 mm Hg in 33% of subjects with exercise-induced PAP increase. In our group of 16 patients with systemic sclerosis (11 subjects with exercise-induced PH and five with PH at rest), RHC showed an elevated pulmonary arterial wedge pressure in 12 (75%) of them. These findings may suggest a latent left-ventricular filling dysfunction. Interestingly, these observations suggest that left-ventricular diastolic dysfunction in patients with connective tissue disease is more frequent than previously believed. Our data indicate that left-ventricular preload reduction using small doses of diuretics may be beneficial for such patients.

In our opinion, Kovacs et al1 are perfectly right that EDE is a useful, noninvasive method to detect systolic PAP increase during exercise in patients with connective tissue disease. However, it should be remembered that RHC still remains the golden standard for assessment of PH.

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DOI: 10.1378/chest.10-1475

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pulmonary hypertension (PH) exists. 2,3 This does not imply that right-sided heart catheterization remains the gold standard for the diagnosis of PH.


Response

To the Editor:

We appreciate the comment by Dr Ciurzyński et al concerning our recently published data (August 2010) on the potential role of exercise Doppler echocardiography (EDE) as a non-invasive screening tool. We also appreciate the agreement of Dr Ciurzyński et al with our main statements and that their unpublished data support our results.

We agree that currently no definition of exercise-induced pulmonary hypertension (PH) exists.2 This does not imply that exercise-induced pulmonary artery pressure (PAP) changes are clinically irrelevant. We need more data providing us with evidence for a reintroduction of a new definition of exercise-induced PH.

We also agree that an exaggerated increase of PAP during exercise may be a sign of early pulmonary vasculopathy and may represent an intermediate stage between a physiologic response and manifest PH. This has already been discussed in a recent article by Tolle et al.4 According to our own results, an out-of-proportion PAP increase at low exercise levels may be especially clinically relevant.5

We also agree that besides pulmonary vasculopathy, a PAP increase during exercise may be triggered by other factors, such as a diastolic dysfunction of the left ventricle. This question is also discussed in our article. According to the unpublished data mentioned by Ciurzyński et al, an elevated pulmonary arterial wedge pressure was found in a very large proportion (75%) of their patients with scleroderma with manifest PAH or exercise-induced PAP increase. We found an elevated pulmonary arterial wedge pressure increase in “just” 33%. This discrepancy may be caused by different methods, different definitions, different patient characteristics, or by chance. A larger prospective study would be necessary to answer the question of how frequently a left ventricular filling dysfunction occurs and on what it depends. The methods applied by Ciurzyński et al are similar to a previous article by Steen et al6 (positive response: increase of at least 20 mm Hg in the right ventricular systolic pressure with exercise), although the data here also show some differences (positive cases 44% in Steen et al vs 17% in Ciurzyński et al).

Despite this variety of the data, the main message remains the same for all the studies, as we also state in the conclusion of our article—that EDE may serve as a useful screening tool for exercise-induced PAP increase, but that right-sided heart catheterization remains the gold standard for the diagnosis of PH.

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Financial/nonfinancial disclosures: The authors have reported to CHEST the following conflicts of interest: Dr Kovacs has received pharmaceutical company grant monies from Actelion Austria GmbH, as well as travel accommodations and speaking honoraria from multiple pharmaceutical companies, including Actelion, GlaxoSmithKline, Astra Zeneca, and Bayer-Schering Pharma. Dr Olschewski received university, European Union, and pharmaceutical industry grant monies and royalties from Actelion, Pfizer, Bayer-Schering Pharma, and GlaxoSmithKline. Dr Olschewski also is a consultant to Unither, Novartis, and Bayer-Schering Pharma, and had speaking activities for Actelion, Bayer-Schering Pharma, GlaxoSmithKline, Pfizer, and Unither.

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DOI: 10.1378/chest.10-1740

REFERENCES


Thoracentesis and Chest Tube Management in Critical Care Medicine

A Multicenter Survey of Current Practices

To the Editor:

Chest tube insertion is commonly performed in the ICU. As with all invasive techniques, adherence to current guidelines and consensus-based practices should reduce potential iatrogenic life-threatening complications. Although recommendations have been published,1 nothing is known about thoracocentesis practices in the ICU.

To investigate these points, a 25-item questionnaire was sent individually to the 634 senior intensivists working in the Ile-de-France in the ICU. To investigate these points, a 25-item questionnaire was sent individually to the 634 senior intensivists working in the Ile-de-France in the ICU. As with all invasive techniques, adherence to current guidelines and consensus-based practices should reduce potential iatrogenic life-threatening complications. Although recommendations have been published,1 nothing is known about thoracocentesis practices in the ICU.

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