with heart and/or lung disease develop PH while others do not is still unknown. It is likely that there are genetic determinants of disease susceptibility that are yet to be elucidated. Whether these mechanisms involve vascular reflexes like the Hemo-Weiler reflex in patients with heart disease, hypoxic pulmonary vasoconstriction in patients with lung disease, or other diverse mechanisms involved in the pathogenesis of PH is unknown.

Sif Hansdottir, MD
Iowa City, IA

Affiliations: From the University of Iowa Carver College of Medicine.

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

Correspondence to: Sif Hansdottir, MD, University of Iowa Hospitals and Clinics, 200 Hawkins Dr, C33GH, Iowa City, IA 52242; e-mail: sif-hansdottir@uiowa.edu

© 2014 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians. See online for more details.

DOI: 10.1378/chest.13-2157

REFERENCES


Systolic BP and Heart Rate in Pulmonary Hypertension

To the Editor:

In a recent issue of CHEST (September 2013), Bersohn et al used the ratio of systolic BP to heart rate (HR) to identify a cohort of patients at high risk in the Registry to Evaluate Early and Long-term PAH [Pulmonary Arterial Hypertension] Disease Management (REVEAL Registry). The work ought to be commended for the identification of two simple bedside clinical parameters to provide prognostic information, which is extremely valuable for clinicians like us. However, we wish to note that it is similar to the shock index (SI), which is the ratio of HR to systolic BP, a variable that provides prognostic information about patients with acute pulmonary embolism (APE). As the authors have the raw data, it should be possible to know if the SI can be calculated in this cohort and if the information is as robust as the index they propose. If it is, we could have a common index that has prognostic value in both acute and chronic pulmonary hypertension. As some cases of APE evolve into chronic thromboembolic pulmonary hypertension, this index may have value in sequential follow-up.

Our unpublished data have revealed that of 35 patients who presented to our district hospital with APE diagnosed by multidetector CT pulmonary angiography, 15 underwent thrombolytic therapy (12 received alteplase and three received tenecteplase). The prethrombolysis SI in the patients submitted to thrombolysis was higher (0.94 ± 0.23) compared with those who were treated with standard anticoagulation using heparin (0.70 ± 0.20). After thrombolysis, the SI calculated using the highest recorded BP and simultaneous HR in the first 24 h decreased to 0.63 ± 0.11 (P < .0001, paired t test). The systolic BP values were not different before and after thrombolysis in the group (125 ± 19 mm Hg vs 126 ± 13 mm Hg, respectively), but HR was significantly decreased postthrombolysis (106 ± 18 beats/min vs 80 ± 15 beats/min, P < .0001). Irrespective of the index used, the two parameters provide indirect information on cardiac output, with tachycardia being a unifying feature of low-output states.

AFFILIATIONS: From the Centro Hospitalar Barreiro Montijo–Internal Medicine (Dr Nobre); and Centro Hospitalar Barreiro Montijo–Cardiology (Dr Thomas).

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.

CORRESPONDENCE TO: Boban Thomas, MD, FCCP, Centro Hospitalar Barreiro Montijo–Cardiology, Barreiro 1900-250, Portugal; e-mail: bobantho@gmail.com

© 2014 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians. See online for more details.

DOI: 10.1378/chest.13-2119

REFERENCES


Response

To the Editor:

We were previously unaware of research using the shock index (SI) in patients with pulmonary embolism, which Drs Nobre and Thomas have brought to our attention in their comments regarding our article. We appreciate their sharing unpublished data about prognostic indicators in acute pulmonary embolism. Because SI = heart rate (HR)/systolic BP (SBP), it is the inverse of the SBP/HR that we found to be a strong predictor of survival and hospitalization in a cohort of 2,830 patients with pulmonary arterial hypertension in the Registry to Evaluate Early and Long-term PAH [Pulmonary Arterial Hypertension] Disease Management (REVEAL Registry).

Therefore, the SI would have exactly the same prognostic value as SBP/HR except that high values would indicate a worse prognosis and low values would indicate a better prognosis. It is gratifying to learn that in patients with acute pulmonary embolism the SBP/HR also correlates with the severity of disease, as suggested by the decision to treat with thrombolytic therapy, and with the response to therapy.

Malcolm M. Bersohn, MD, PhD
Shelley Shapiro, MD, PhD
Los Angeles, CA