Reviewer's report

Title: Novel Computed Tomographic Chest Metrics to Detect Pulmonary Hypertension

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Reviewer: George Washko

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In this manuscript “Novel Computed Tomographic Chest Metrics to Detect Pulmonary Hypertension”, Chan and colleagues retrospectively examine CT and RHC data on 101 hospitalized patients with a range of clinical disorders. Using both objective and subjective assessments of the CT images, they identified a series of structural measures (i.e. vascular diameter, ventricular free wall thickness, etc.) which were correlates to data obtained using more invasive techniques. Based upon these results, Chan and colleagues suggest that CT assessments of cardiovascular structures may be a simple and safe screening methodology for the presence of pulmonary hypertension.

Overall, this manuscript is well written and presents metrics of remodeling that can be replicated at most if not all clinical institutions. The results are interesting and as the authors suggest, further investigation is warranted. Potential limitations to this investigation include the non-standardized CT acquisition protocol (contrast vs no contrast) and non ECG gated images.

Major Comments:

1. These CT scans were non-ECG gated. While one may argue that motion artifact and volume averaging should introduce a bias to the measures but not reduce their precision, is that really the case for assessments of the heart? One may reasonably expect that heart rate would affect measures of cardiac structure and may even lead to greater overestimation of structure size. If the sickest patients are also the ones with the PH, could the results of measures of cardiac size simply be a measure of HR induced effects on the images rather than a change in the structures themselves? Can the authors address this concern using additional clinical data such as resting pulse?

2. This investigation included the use of both I+ and I- CT scans. Did the authors use all of the scans, or just the I+ images when assessing cardiac structures? Can one obtain dependable measures of wall thickness using I- scans? Please clarify.

3. In the discussion, the authors mention that the utility of CT screening for disease lies in the argument that early detection leads to early treatment and reduced remodeling when presumably leads to reduced morbidity and mortality. To support this statement, an estimate of 2.8 years median survival in untreated PH is presented. Is this true of all forms of PH, i.e. due to ARDS, CHF, COPD, hypoventilation, etc or just for idiopathic PH? Generally, the other forms of PH
such as that found in subjects with COPD is not well enough studied to support such a median survival time. Please clarify or further substantiate.

4. Interestingly, the authors mix PH which is presumably due to both venous (primarily LV dysfunction) and arterial disease. Surprisingly, these measures still where useful in disease detection. Further, the authors mix acute conditions (ARDS) with chronic conditions and still see statistical significance. Please clarify or hypothesize why measures of ventricular wall thickness would be pertinent in both acute and chronic conditions as well as those with both venous or arterial PH. Could CT be used to discriminate arterial from venous disease or acute from chronic disease?

5. The authors include both spontaneously and mechanically ventilated patients in this investigation. As the authors point out, it is a tenuous assertion to include both in the same hemodynamic investigation. The authors should include a secondary analysis of only those subjects who are not mechanically ventilated to determine if their findings are robust. There are not enough subjects to make similar assessments of the mechanically ventilated cohort.