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A Step Towards Hemodynamic Assessment with CT Angiography

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In this era of multi-modality imaging, no single noninvasive approach always takes the prize. Each technique has its strengths and weaknesses in terms of diagnostic information provided, and attaining the goal of optimal patient care requires a detailed understanding of both the pertinent clinical questions to be answered and how these questions can be best addressed given the technical details of each modality. Coronary CT angiography (CCTA) has found an increasingly important role in the care of patients with diseases of the cardiovascular and respiratory systems, in large part due to its unmatched spatial resolution which enables accurate assessment of coronary anatomy and pathology. A common perception, however, is to view CCTA as incapable of providing physiologic or hemodynamic data.

In truth, however, the capability provided by technological development in CT “provides a very powerful new tool for increasing the knowledge and understanding of the physiology of these organ systems and the mechanisms of environmental or disease processes which affect their structure or function in health or disease.” This idea is not new. In fact, this prescient remark was made in the 1970s by the late Earl Wood¹, then president-elect of the American Physiological Society, and his colleagues at the Mayo Clinic Biodynamics Research Unit, in anticipating the future uses for their yet-to-be-built Dynamic Spatial Reconstructor, which would become the world's first-ever cardiac CT scanner.² While efforts towards advancing cardiac CT have properly centered on its strengths as an anatomic probe, but that is not to say that CT cannot be used to characterize cardiovascular physiology. Indeed, in this issue of *JCCT*, Dusaj et al demonstrate the hemodynamic potential of CCTA, proposing methodology to quantitatively estimate right ventricular pressure based on CCTA-derived measures of vena caval contrast reflux column length and distensibility.

Several prior studies set the stage for this work. Early work in echocardiography demonstrated reflux of microbubble contrast into the inferior vena cava (IVC) and hepatic veins to be a non-specific sign of tricuspid regurgitation.³ Similarly, studies of various CT examinations showed that IVC contrast reflux was associated with pulmonary hypertension, right ventricular systolic dysfunction, and tricuspid regurgitation.^{4–8} Groves et al showed good correlation between CT and echocardiography in semi-quantitatively grading the

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severity of tricuspid regurgitation.⁴ CT protocols may confound this finding, however, as Yeh et al found that high contrast injection rates (greater than 3 mL/sec) along with tricuspid regurgitation, pulmonary hypertension, and right ventricular systolic dysfunction were independent predictors of IVC and hepatic vein contrast reflux.⁵ Another study, by LaBounty et al, showed no association between IVC contrast reflux and tricuspid regurgitation in CCTA when compared to transesophageal echocardiography.⁹ Dusaj et al expand on the proposed association between IVC contrast reflux and right-sided heart disease to derive a quantitative estimate of right ventricular systolic pressure from CCTA measurements. As such, this paper represents the first effort in advancing CT to the next logical level in characterizing right heart physiology, moving from semi-quantitative characterization of disease severity to numerical estimates of right-sided pressures.

Specifically, in this retrospective study, transthoracic echocardiography and retrospectively-gated CCTA were reviewed for 32 patients. Prior to CCTA, patients were given metoprolol tartrate and sublingual nitroglycerin. Measurements of IVC contrast reflux column length and SVC distension from CCTA were compared to transthoracic echocardiography measurements of tricuspid regurgitation jet velocities and IVC sniff response ratio, respectively. There were no other statistically significant correlations between CCTA and echocardiography findings including comparisons between IVC and SVC distension to tricuspid regurgitation jet velocity. IVC contrast reflux column length and SVC distension were measured using standard subcostal views and short axis views at the level of pulmonary artery bifurcation, respectively. Intra- and inter-observer variability of IVC contrast reflux column length and SVC distension was adequate. A strong correlation was determined between IVC contrast reflux column length and tricuspid regurgitation jet velocity while a moderate correlation was found between SVC distention ratio and IVC sniff response ratio. From these CCTA measurements, right ventricular systolic pressure was estimated using a modified Bernoulli equation. In this equation, tricuspid regurgitation jet velocity was replaced by the fitted linear equation including the IVC contrast reflux column length from the previous comparison while right atrial pressure was replaced by a semi-quantitative estimate derived from the SVC distention ratio.

The study is not without weaknesses. A primary concern, acknowledged by the authors, is the use of echocardiographic estimates as the reference standard rather than the gold standard of right heart catheterization. For validation, this new technique needs to be compared to invasive, direct measurements that right heart catheterization provides. The patient population in this study was quite small and also limited by only including a small proportion of patients having right atrial or ventricular enlargement, pulmonary hypertension, right ventricular systolic dysfunction, or any degree of tricuspid regurgitation. Studying additional patients with right-sided heart disease is needed to validate the correlation determined between IVC contrast reflux column length and tricuspid regurgitation jet velocity and the derived equation for right ventricular systolic pressure estimation. Another issue regarding the comparison between the measurements from CCTA and transthoracic echocardiography was the delay between these examinations. Although no changes in clinical status were noted in any of the patients, it is difficult to state that the patients' hemodynamics were unchanged given the time between exams, with a mean (range) of 10 (0 to 59) days, and the routine use of beta blockers and nitrates prior to CCTA but not echocardiography.

An additional limitation of the study is the model-building strategy employed for developing an estimator of right ventricular systolic pressure. The only parameters considered for inclusion in this model were contrast reflux column length and a dichotomized SVC distension ratio, the latter serving as an approximation of right atrial pressure. In building an optimal multivariate model, the statistical literature^{10–11} supports considering for inclusion

covariates which don't quite meet significance on univariate analysis, e.g. consideration of variables with univariate p values <0.20 .¹⁰ Could estimation of RVSP be improved by incorporating, for example, pulmonary artery diameter into the prediction model? Moreover, no regression diagnostics were performed to identify potential outlier data points which could distort the RSVP estimation model presented.¹²

Despite these limitations, this paper by Dusaj et al truly represents a significant advance, and sets the stage for further investigations that develop more refined models to estimate right-sided hemodynamics from CT parameters. Because of this contribution, this work “takes the prize”, as a recipient of SCCT's fourth annual Young Investigator Award.

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