Coronary slow flow (CSF) is a well-recognized clinical entity, characterized by delayed opacification of coronary trees without any evidence of epicardial obstructive disease. This phenomenon was originally identified in 1972 by Tambe, et al.1) The overall incidence of CSF is 1-7% in patients who undergo diagnostic coronary angiography for clinical suspicion of coronary artery disease. Although this phenomenon is well known to interventional cardiologists, the exact pathophysiologic mechanisms are still unclear. CSF is most commonly observed in young smokers with acute chest pain.2) It does not represent a simple angiographic curiosity, but rather CSF has direct clinical implications such as recurrent chest pain, life-threatening arrhythmias, and sudden death.3,4) There is a strong association between metabolic disorders and CSF. Higher low-density cholesterol, fasting glucose, and body mass index values were more frequent in patients with CSF than those without CSF.5) Consistently, impaired glucose tolerance6) and insulin resistance7) correlated with the occurrence of CSF. However, the pathogenesis of CSF is still unclear. There are some typical histopathological features associated with CSF. Previous studies reported fibromuscular hyperplasia, medial hypertrophy, and endothelial edema in coronary microvessels with consequent decrease in luminal diameter that led to functional obstruction and CSF.8) Coronary endothelial dysfunction is also considered to play an important role in CSF.9) Furthermore, elevated aortic pulse pressure was observed in patients with CSF compared to those without CSF.10) Finally, impaired coronary flow reserve might be involved in the occurrence of CSF.11)

In this issue of International Heart Journal, Shi and colleagues examined and compared arterial compliance and layer-specific longitudinal and circumferential strain assessed by speckle-tracking echocardiography in 120 patients (70 CSF and 50 control patients) who underwent invasive coronary angiography.12) Among the 70 patients with CSF, 68 (97%) showed CSF in the left anterior descending artery, 66 (94%) in the circumflex artery, and 63 (90%) in the right coronary artery. Indexed arterial compliance was significantly lower in patients with CSF, suggesting greater pulsatile arterial load in the CSF group. Importantly, although layer-specific global longitudinal strain (GLS) showed a decreasing gradient from the endocardium to the epicardium in both the control and CSF groups, GLS-endo and GLS-mid were significantly lower in patients with CSF compared to controls.

Similar results have been reported in other small studies. Kemaloglu, et al. demonstrated significantly impaired left ventricular (LV) and right ventricular strain in patients with CSF using 3-dimensional speckle tracking echocardiography in 80 patients who underwent coronary angiography.13) Barutcu, et al also showed significantly impaired LV twist, torsion and apical rotation in the CSF group compared to a control group with normal coronary arteries.14) Although these studies found consistent results, a number of issues still need to be clarified. No study has evaluated whether CSF independently impairs LV contractility because of the cross-sectional study designs and limited numbers of patients. As we mentioned above, patients with CSF had a higher prevalence of cardiovascular risk factors which may affect the association between CSF and impaired LVGLS. Furthermore, there is limited data on therapeutic intervention and prognosis in patients with CSF. Future studies are warranted to investigate the underlying pathophysiological mechanisms linking CSF and impaired LVGLS and to evaluate whether medications such as statins15,16) and dipyridamole,17) which might be possible therapies for CSF, improve subclinical LV dysfunction.

Disclosures
Conflicts of interest: There is no conflict of interest or financial disclosure pertinent to the content of the manuscript.
References


