Hypertension is an important risk factor for cardiovascular disease (CVD), end-stage renal disease, and mortality. In early 1960s', randomized controlled trials demonstrated the value of treating high diastolic blood pressure (DBP) and subsequently high systolic BP (SBP). Therefore, DBP was originally the main focus to evaluate the risk in association with high BP and to set the treatment goal, but the center of gravity has gradually moved to SBP. Recently, SPRINT demonstrated that intensive SBP lowering improved CVD outcomes and all-cause mortality in adults at high risk for CVD events. Indeed, low baseline DBP was also associated with increased risk of CVD, and there was no evidence that the benefit of the intensive SBP lowering differed by baseline DBP.

There is limited RCT evidence regarding optimal BP targets in the CKD population, but a line of evidence from observational studies suggests a "U-shaped" relationship between BP and adverse outcomes in CKD. In addition, the clinical significance of various BP components, including SBP, DBP, and pulse pressure (PP), has been investigated in CKD, and they are unlikely to have the same influence on various clinical outcomes. Among participants of CRIC trial with stage 4 and 5 CKD, there was an independent association between higher SBP, DBP, and PP with the risk of atherosclerotic CVD, whereas only higher PP was independently associated with a greater risk of heart failure.

In this session, we’re going to have a glimpse at the necessity that we should take other BP parameters except SBP to get an optimal outcome in CKD patients.