also positively correlated with the presence of knee beam hardening artifact. Compared to standard setting, the use of R2 settings decreased sensitivity (0.79 [95% CI: 0.65-0.88] versus 0.90 [95% CI: 0.78-0.96]) and increased specificity (0.86 [95% CI: 0.71-0.93] versus 0.83 [95% CI: 0.47-0.77] (p=0.001).

Conclusion: Applying a ratio of 1.28 and a minimum attenuation of 170 Hu (R2 settings) in DECT post-processing eliminates the majority of the artifacts located on the lower limbs, particularly the clumpy artifact and the beam hardening artifact.

REFERENCES:
[1] Neogi T, Jansen TLTA, Dalbeth N, Fransen J, Schumacher HR, Berendse D, et al. 2015 Gout classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. Ann Rheum Dis. 2015;74:1789-98.

Table 1. Mean (SD) sUA Levels Following Once-Daily Oral Doses of AR882.

| Treatment | N | 0       | 6       | 12      | 24      |
|-----------|---|---------|---------|---------|---------|
| Baseline  | 30| 8.8 (1.1)| 8.8 (1.1)| 9.0 (1.0)| 8.9 (1.1)|
| 25 mg     | 9 | 6.1 (0.4)| 5.6 (0.4)| 5.6 (0.3)| 5.8 (0.4)|
| 50 mg     | 28| 4.7 (0.9)| 4.2 (0.9)| 4.3 (0.8)| 4.7 (1.0)|
| 75 mg     | 8 | 3.7 (1.1)| 3.2 (0.9)| 3.2 (0.8)| 3.6 (1.0)|
| 50 mg (normal renal function) | 17 | 4.8 (1.1) | 4.3 (1.1) | 4.4 (0.9) | 4.8 (1.2) |
| 50 mg (mild impairment) | 11 | 4.5 (0.6) | 4.1 (0.6) | 4.3 (0.5) | 4.6 (0.6) |

 blossoms and early season has utility in the treatment of patients with
so far from being elucidated. The conflicting evidence has made it difficult
tify the extent of the cardiovascular risk to patients with gout.

Objectives: To describe the incidence and prevalence of cardiovascular disease in gout, compare these results with non-gout controls.

Methods: PubMed, Scopus and Web of Science were systematically searched in January 2021 for studies reporting prevalence of any cardiovascular disease in a gout population. Studies with non-representative sampling, where a cohort had been used in another study, small sample size (< 100) and where gout could not be distinguished from other rheumatic conditions were excluded. Sample size, prevalence of the investigated cardiovascular disease, definition of gout and cardiovascular disease, demographic data, data source and any comparisons with non-gout controls were extracted from each study. Where prevalence data was reported in ≤3 cohorts meta-analysis was performed including at the meta-analysis level.

Results: Of the 6164 titles identified, 105 full texts were assessed for eligibility with 30 included in the review, producing a gout population of 1,125,988. Pooled prevalence estimates were calculated for six cardiovascular diseases: heart failure (8.73%; 95% confidence interval (CI), 2.85 – 23.76%), cerebrovascular accident (4.27%; 95% CI, 1.83 – 9.67%), myocardial infarction (2.82%; 95% CI, 1.58 – 5.01), venous thromboembolism (2.03%; 95% CI, 1.22 – 3.43), hypertension (63.94%; 95% CI, 24.51 – 90.64) and cardiovascular mortality (4.75%; 95% CI, 3.56 – 6.31). Twenty studies reported comparisons with non-gout controls, illustrating an increased risk in the gout group across all cardiovascular diseases, particularly for myocardial infarction.

Conclusion: Cardiovascular diseases are more prevalent in patients with gout and should prompt vigilance from clinicians to the need to assess and stratify cardiovascular risk. These results are in line with other studies which have shown an increased cardiovascular risk for sufferers of hyperuricaemia, highlighting the need for future research to explain this finding. There are limited studies in the