Achieving adequate adenosine induced vasodilatation is paramount in the interpretation of myocardial perfusion imaging. Current methods of monitoring vasodilatation rely on reported side-effects during the adenosine infusion, or analysis of the myocardial perfusion reserve via quantitative dynamic PET imaging. It has previously been observed in cardiac magnetic resonance imaging [1] that the difference in perfusion of the spleen at stress and rest is a potential marker for adequate stressing of the heart by adenosine. This work explores the use of splenic ‘switch-off’ in \(^{82}\)Rb myocardial perfusion imaging to evaluate adequate vasodilatation during adenosine stress.

A total of 46 patients referred for a dynamic \(^{82}\)Rb PET myocardial perfusion study were retrospectively analysed. Patients were included if they had normal patterns of perfusion and no previous history of CAD. Of these, 39 (Group 1) demonstrated side-effects consistent with adequate adenosine vasodilatation and 7 (Group 2) did not report side-effects.

All patients had their myocardial blood flow and perfusion reserve (MPR) measured as per routine clinical protocol, corrected for blood pressure and heart rate using the rate-pressure product (RPP). Patients with significant motion during their dynamic acquisition were excluded.

To investigate the time point during the \(^{82}\)Rb infusion at which to assess the splenic perfusion, an in-house Matlab script was developed to produce dynamic uptake curves for a 2cm volume of interest (VOI) within the spleen, see Figure 1.

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\text{SUR} = \frac{C_{\text{REST}}}{C_{\text{STRESS}}} \quad [1]
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Where \(C_{\text{REST}}\) and \(C_{\text{STRESS}}\) represent mean activity concentrations. Differences in the SUR and MPR between Group 1 and Group 2 were assessed using the Mann-Whitney U test for non-parametric data.

There is a clear separation in patients with normal perfusion between those who reported side effects, and those who did not, Figure 2. There was a significant difference in the median SUR between those with (1.58 [IQR 1.35-1.67]) and without (1.03 IQR [0.96-1.09]) side effects, \(p<0.001\). The SUR correlated well with MPR, indicating splenic uptake decreases with enhancement of stress blood flow.

Of those patients not reporting side effects with a low SUR, one was repeated due to the possibility of caffeine intake (\(\text{O}\)) acting as an antagonist to adenosine, with the repeat (\(\text{‡}\)) showing an increase in both the MPR (1.06 to 3.34) and SUR (1.05 to 1.51). Another patient (\(\text{O}\)) who was found to have probable balanced multivessel ischaemia, due to significant calcification in the LMS and LAD, had a high SUR (1.83), but abnormal MPR (0.81).

These two patients indicate that the SUR could potentially help differentiate between those patients with a low MPR due to multi-vessel disease and those who have not been adequately stressed due to the presence of adenosine antagonists, although further investigation is required.

**Conclusions**

- All patients with normal perfusion and reporting side-effects during their adenosine stress had an SUR of greater than 1.2.
- Including patients with and without side-effects, there was a weak correlation between MPR and SUR.
- The SUR could be useful in cases where differentiation between adenosine antagonists (e.g. caffeine) and true multi-vessel disease is not possible using conventional methods.

**References**

1. Manisty et al. (2014). J Cardiol Mag Res. 16(Suppl 1):O1