had isolated microvascular angina, 25 (17%) had isolated vasospastic angina, 31 (20%) had both (MVA & VSA) only 17 (11%) had non-cardiac chest pain. Myocardial bridging of coronary artery was found in 22 (15%). Multivariate predictors of MVA included typical angina, inducible ischaemia but traditional cardiovascular risk factors were not associated. Smoking and age were independent predictors of VSA.

**Conclusion** The majority of patients with symptoms and/or signs of ischemia and no obstructive disease have a diagnosis of microvascular and/or vasospastic angina. Traditional cardiovascular risk scores have limited discrimination for disorders of coronary vasomotion.

**Conflict of Interest** Nil

---

**Abstract 51 Table 1** Clinical characteristics of the forty patients according to whether they were or were not shown the animation before their procedure

<table>
<thead>
<tr>
<th></th>
<th>Not shown Animation (n=20)</th>
<th>Shown Animation (n=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>17</td>
<td>13</td>
</tr>
<tr>
<td>Female</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Angiogram</td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>PCI</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Elective</td>
<td>20</td>
<td>19</td>
</tr>
<tr>
<td>Previous Angiogram</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>English Speaker</td>
<td>11</td>
<td>14</td>
</tr>
</tbody>
</table>

*p value not significant for any differences in variables between two groups*

---

**Abstract 51 Figure 1** Patient understanding of the procedure in the animation and no animation groups.
from the novel coatings, with 45 ±1.8% and 87 ±3.2% of radicals scavenged at 8 and 24 h respectively (one way ANOVA, n=3, p<0.05). Antioxidant activity was sustained over 5 days. HUVECs were cultured onto coated stainless steel strips and imaged using immunofluorescence to assess their attachment and viability.

To model pro-inflammatory and pro-oxidative stress conditions, HUVECs were treated with IL-1β, which is known to be elevated in both conditions. ROS generation in HUVECs in response to IL-1β (3–10ng/ml; 1–6h) stimulation was assessed using a DCFDA (2',7'-dichlorofluorescin diacetate)-based assay kit (Abcam, UK). IL-1β stimulated ROS production in a time and dose dependant manner with optimal effects at 3 ng/ml after 6 hours (3.3 ±0.73-fold increase between stimulated and unstimulated cells, one way ANOVA, n=3, p<0.05). Parallel assessment using quantitative immunoblotting of CaMKIIβ in HUVECs showed the enzyme was expressed and that activation (via phosphorylation or oxidation) can be detected. Increased phosphorylation of P65 in response to IL-1β (10ng/ml; 15 min-6h) stimulation was also detected, indicating increased pro-inflammatory signalling.

In conclusion, we have established an effective cell-based model for mimicking the oxidative stress that can occur following stent placement and identified CaMKIIβ as a target protein. This approach will be applied to other more physiologically relevant cell types and may be used to examine the desired therapeutic effects of novel stent coatings in vitro.

Conflict of Interest N/A

53 VIRTUAL (COMPUTED) FFR AND VIRTUAL CORONARY INTERVENTION (VCI) VS ANGIOGRAPHY FOR GUIDING PCI: A VIRTUAL STUDY

1Rebecca Gosling*, 2Paul Morris, 3Patricia Lawford, 4Rodney Hose, 5Julian Gunn.
1University of Sheffield, 2Department of Infection, Immunity and Cardiovascular Disease, University of Sheffield

Abstracts

Introduction Using fractional flow reserve (FFR) to guide percutaneous coronary intervention (PCI) improves outcomes and reduces costs. In FAME, FFR guidance reduced the total length of stent per patient from 52mm to 38mm and the number from 2.7 to 1.9(1), yet FFR is currently used in <10% of cases (2). Angiography-based virtual (v) FFR solutions permit less invasive physiological assessment and lend themselves to virtual coronary intervention (VCI) (Figure 1). VCI has been shown to predict the response to PCI with a high degree of accuracy(3). In this study, we sought to determine the potential impact of vFFR and VCI on real world stenting.

Methods Patients who had undergone PCI without FFR guidance were identified from the Sheffield archive. A 3D reconstruction of the diseased artery was generated from the angiogram. Baseline vFFR was calculated using computational fluid dynamics (CFD) analysis(4). If vFFR was <0.80, VCI was performed(3). Three PCI strategies were modelled. First, the actual PCI procedure was replicated. Second, the FFRmax was determined; the minimal amount of stenting to achieve the best possible FFR(5). Third, the optimal strategy was determined; the minimal amount of stenting to achieve a post VCI FFR >0.90 (6). For each strategy, the total number and length of stent per patient was determined and compared to the actual procedure, which was conducted in the normal way, guided by the angiogram alone.

Results Forty-three patients (56 vessels) were studied. Mean vFFR pre-PCI was 0.74±0.16. Twenty-four (43%) vessels had a baseline FFR >0.80. For the actual procedure, mean post-PCI vFFR was 0.90±0.09. The number of stents per patient was 1.4±0.6. Total stent length per patient was 29±15mm. Mean FFRmax was 0.92±0.07. FFRmax was 0.02±0.03 higher than the corresponding measured post-PCI FFR. When the virtual procedure was planned to achieve FFRmax, the number of stents per patient was 0.9±1.0 (p=0.003). Total stent length per patient was 22±27mm (p=0.04). When the virtual procedure was planned to achieve a post VCI FFR >0.90, the number of stents per patient was 0.93±1.02 (p=0.002). Total stent length per patient was 20±25mm (p=0.01).

Conclusion In our cohort, 43% of vessels had a vFFR >0.80 suggesting PCI could have been avoided. Using vFFR and VCI to plan PCI led to a significant reduction in the total number and length of stents recommended per patient. Further work on a larger cohort is required to determine if these findings would translate to improved clinical outcomes.

Figure 1: The 3D reconstruction of the artery is displayed and the operator marks the location where they wish to deploy a stent identified by the red and the blue markers (A). The operator can adjust the radius of the desired virtual stent in the box labelled ‘stent size’. The length can be altered by moving the position of the red and blue dots. In the example shown, a 3.0mmx20mm virtual stent has been inserted. The virtually stented artery is shown overlaying the original vessel in panel B. Reproduced from JACC: Cardiovascular Imaging (3) under creative commons license CC BY 4.0

Conflict of Interest None

Abstract 53 Figure 1