variability (HRV) albeit with considerable methodological limitations, particularly with respect to temporal resolution. However, recent advances have allowed the measurement of a novel non-invasive validated measure of efferent vagal activity from the brainstem, known as cardiac vagal tone (CVT). CVT is measured on a linear vagal scale (LVS) where 0 represents full atropinization and has improved temporal resolution compared to HRV. CVT is increasingly being utilised in a diverse array of GI research (3.4.5). However, its normal values and reproducibility are, to date, incompletely understood. The aim of this study was to address these knowledge gaps.

**Methods** 120 healthy subjects (68 males, median age 29 years, range 19–55 years) were studied in a temperature controlled, constantly lit, quiet laboratory. After attachment of CVT recording equipment (Neuroscope), 20 minutes of CVT data (resting/no stimulation) was acquired. 30 subjects, selected at random, were restudied after 1 year. Reproducibility was assessed using a two-way, random effects, single measure intra-class correlational coefficients (ICC) model and Bland Altman plots.

**Results** The mean CVT was 8.2 LVS with a standard deviation of 3.0. Thus, the normal range (mean +/- 2 standard deviations (SD)) for CVT based on this data is therefore 2.2 LVS to 14.2 LVS. Age correlated negatively with CVT (r = -0.36, p < 0.0001) but there was no discernable effect of gender, body mass index or ethnicity. The ICC for CVT was 0.81 (95% confidence interval 0.64–0.91), indicating excellent reproducibility. Figure 1 shows the Bland-Altman plot that demonstrate that 29 out of the 30 measurements lie within +/- 2 SDs of the differences between measurements suggesting that there was no bias or systematic error and that the parameter of CVT is reproducible at a period of 1 year.

**Conclusion** The normal range for CVT should be considered to be 2.2 – 14.2 LVS. CVT is a reproducible measure over the period of 1 year. Future research utilising CVT should refer to these values

**Disclosure of Interest** None Declared

**REFERENCES**

**Abstract PTU-121 Figure 1**

**Conclusion** The normal range for CVT should be considered to be 2.2 – 14.2 LVS. CVT is a reproducible measure over the period of 1 year. Future research utilising CVT should refer to these values

**Disclosure of Interest** None Declared

**REFERENCES**

**Disclosure of Interest** None Declared

**REFERENCE**

**PTU-123**

**FACTORS THAT ANTICIPATE CLINICAL/ThERAPEUTIC OUTCOMES TO PRUCALPOPRIDE**

**Introduction** Chronic constipation (CC) is a prevalent disorder that has a significant negative impact on quality of life. Traditional management has focused on lifestyle measures and laxative. Prucalopride, a selective high affinity 5-HT3 receptor agonist, has been demonstrated to an effective treatment of severe CC. However, its efficacy in secondary care and factors that predict clinical response are incompletely understood. Our aim was thus to identify baseline factors that may predict positive clinical outcomes in patients taking prucalopride for CC.

**Methods** A single centre, prospective open label trial was undertaken in patients with primary and secondary CC, defined as less than 2 spontaneous complete bowel movements (SCBM) per week, who were commenced on prucalopride. Validated questionnaires

**Disclosure of Interest** None Declared

**REFERENCE**
1. A D Farmer, P Harvey, D Haldar, N Quraishi, Q Aziz. Wingate Institute of Neurogastroenterology, Barts & The London School of Medicine, London; 2Gastroenterology, Shrewsbury & Telford NHS Trust, Telford, UK

**Disclosure of Interest** None Declared

**REFERENCE**

**Disclosure of Interest** None Declared

**REFERENCE**
1. A D Farmer, S J Coen, N M Kano, Q Aziz. Wingate Institute of Neurogastroenterology, Barts & The London School of Medicine, London, UK

**Disclosure of Interest** None Declared

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1. A D Farmer, P Harvey, D Haldar, N Quraishi, Q Aziz. Wingate Institute of Neurogastroenterology, Barts & The London School of Medicine, London; 2Gastroenterology, Shrewsbury & Telford NHS Trust, Telford, UK

**Disclosure of Interest** None Declared

**REFERENCE**
1. A D Farmer, S J Coen, N M Kano, Q Aziz. Wingate Institute of Neurogastroenterology, Barts & The London School of Medicine, London, UK
were used to assess the severity of symptoms (patient assessment of constipation symptoms (PAC-SYM)), somatic symptoms (patient health questionnaire (PHQ-12-SS)) and the personality trait of neuroticism (big five inventory-neuroticism scale (BFI-N)). At follow up, clinical response was defined as the proportion of patients achieving 3 or more SCBM per week.

**Results** 64 patients (59 female, mean age 48.3 years, range 19–83) had a mean SCBM per week of 1.6 (range 0.5–2). At a mean follow up of 4.6 weeks (range 4–8) 40/64 (62.5%) patients achieved clinical response. 8/64 (12.5%) did not tolerate treatment due to side effects. In an intention to treat analysis, mean SCBM per week increased from 1.6 to 3.2 (p = 0.01) with mean PAC-SYM scores reducing from 27.7 to 20 (p = 0.001). Logistic regression analysis demonstrated that BFI-N (odds ratio 8.7, 95% confidence interval (CI), 1.99–64, p < 0.01) and slow transit constipation (STC) (odds ratio 1.4, 95% CI, 1.20–2.1, p < 0.01) were independently associated with positive treatment outcomes.

**Conclusion** Prucalopride is a useful, generally well tolerated, treatment for the management of CC in secondary care. These data suggest that efficacy could be enhanced by targeting patients with STC and in those who are more neurotic. Further work is now warranted to confirm these findings in a larger cohort of patients.

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**PTU-125**

**THE ASSOCIATION OF THE JOINT HYPERMOBILITY SYNDROME WITH FUNCTIONAL GASTROINTESTINAL DISORDERS – AN INTERESTING NEW FINDING THAT MAY EXPLAIN AETIOLOGY**

**References**

**Introduction** Functional Gastrointestinal Disorders (FGID) are common, but their cause is unknown. Joint hypermobility syndrome (JHS) is a common non-inflammatory connective tissue disorder characterised by joint hyperflexibility. It is associated with gastrointestinal (GI) symptoms, in particular unexplained symptoms. The association between JHS and FGID has never been studied.

**Methods** A nested case control study in patients aged 18–70 attending secondary care was performed. 694 consecutive new referrals to GI clinics were assessed for JHS using the Brighton criteria, prior to their outpatient consultation. Subsequent investigation by their gastroenterologist led to a diagnosis that was functional, organic or gastroesophageal reflux (GOR); the latter were excluded due to the mixed aetiology of reflux. The control group consisted of 92 patients referred to secondary care for non-GI symptoms–those with diabetes, pregnancy, neuromuscular disorders or inflammatory arthritis were excluded. Controls were similarly assessed for JHS. JHS prevalence was compared in patients with FGID, organic GI disorders, and controls.

**Results** Of the 694 GI patients, 26 had GOR and 52 had not received a diagnosis–these were excluded. Thus 616 GI patients were included in the study. 363 had FGID, 253 had organic disorders. There were no significant age or gender differences between FGID and controls (age: 40.3 ± 0.69 vs. 42.7 ± 1.5%, 64% vs. 67% females). Compared to FGID patients, organic patients were older (43.9 ± 0.92 vs 40.3 ± 0.69, p=0.002) and less likely to be female (54% vs 64%, p=0.008). The prevalence of JHS in FGID patients in secondary care was 40.5%. This was significantly higher than in organic GI patients (26.9%, p=0.000) and in controls (25%, p=0.006). Even after adjusting for age and gender differences, JHS was significantly associated with FGID (p=0.005).

**Conclusion** This is the first study that demonstrates a strong association between JHS and FGID, as compared to both organic GI and non-GI conditions. This suggests a potential connective tissue aetiology for 40% of FGID patients in secondary care. Furthermore, the high prevalence of JHS in FGID suggests that this common diagnosis is often overlooked. Our results have implications for future FGID research and efforts must now be focused to determine the mechanism of symptoms and identification of appropriate treatments for this subgroup of patients.

**Disclosure of Interest** None Declared

**PTU-125**

**JOINT HYPERMOBILITY IS A RISK FACTOR FOR OSESOPHAGEAL HYPERSENSITIVITY**

**Disclosure of Interest**

**Introduction** Generalised joint hypermobility (HM) refers to an increased flexibility of the joints which is present in 5–17% of the population, and is assessed by clinical examination or validated hypermobility questionnaire. HM is associated with increased incidence of somatic pain and hypersensitivity, but its relationship with visceral hypersensitivity is unknown. Gastro-oesophageal reflux symptoms occur in over 50% of hypermobile patients. We hypothesised that we would observe a higher prevalence of HM in patients with hypersensitive oesophagus compared to patients with either erosive (GERD) or non erosive (NERD) reflux disease, functional heartburn, or a healthy control group.

**Methods** A cross sectional study of patients attending our GI physiology unit for investigation of reflux symptoms between Jan 2010 and March 2012 was undertaken. Patients completed the validated joint hypermobility questionnaire; scores ≥2 out of 5 represented HM (Hakim 2008). Information from gastroscopy and physiology testing was used to determine whether patients had GERD, NERD, hypersensitive oesophagus, or functional heartburn. Patients who were on PPI were excluded. Hypermobility questionnaire data from a group of 250 healthy volunteers was obtained from another study.

**Results** 457 (59% female, age range: 15–79) patients with complete data were included. HM was present in 79 (17%) patients. The prevalence of HM was highest in patients diagnosed with hypersensitive oesophagus (31%) and lowest in NERD (15%)-see table 1. The prevalence of HM was significantly higher in hypersensitive oesophagus (31%) compared to the combined prevalence in other reflux diagnoses (16%), p = 0.009, and to the healthy control group (18%), p = 0.04.

**Abstract PTU-125 Table 1**

<table>
<thead>
<tr>
<th>Group</th>
<th>Total number</th>
<th>Number with HM (score ≥ 2) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GERD</td>
<td>53</td>
<td>11 (19%)</td>
</tr>
<tr>
<td>NERD</td>
<td>233</td>
<td>35 (15%)</td>
</tr>
<tr>
<td>Hypersensitive oesophagus</td>
<td>49</td>
<td>15 (31%)</td>
</tr>
<tr>
<td>Functional heartburn</td>
<td>122</td>
<td>20 (16%)</td>
</tr>
<tr>
<td>Healthy volunteers</td>
<td>250</td>
<td>45 (18%)</td>
</tr>
</tbody>
</table>

**Conclusion** 17% of patients with reflux symptoms severe enough to warrant physiology investigation have HM, which is similar to the prevalence in healthy controls. The prevalence of HM in patients...