Weighg loss greater than 6% independently correlates with reduced survival in patients with congestive heart failure (CHF). Cardiac cachexia is a gradual and graded process, with wasting affecting muscle, bone, adipose tissue and the heart. Reduced appetite has been proposed as an important factor for weight loss caused by cardiac cachexia.

The mechanism of cardiac cachexia remains largely unclear. Gastric myoneural inhibition and gastrointestinal hypomotility with delayed gastric emptying is commonly observed, and gut hormones may also play a role, because the anorexia is often characterised by a premature feeling of fullness and loss of hunger. The gut hormone ghrelin stimulates hunger, increases food consumption, and was previously shown to be raised in patients with cardiac cachexia and CHF. Ghrelin is produced from the stomach and usually rises in anticipation of a meal and then falls after the ingestion of nutrients.

The gut hormone PYY3–36, acting as a terminator of hunger, is produced by the L cells in the distal gastrointestinal tract; it reduces appetite and 24 hour food intake by binding to the neuropeptide Y2 receptor in the hypothalamic arcuate nucleus.

Severe pulmonary hypertension results in a low cardiac output and neurohumoral activation. Patients frequently report loss of appetite and weight, and since it occurs in a young population they have little co-morbid disease, which itself might affect appetite. This study aimed to evaluate dynamic PYY and ghrelin responses to a standard meal in control subjects and patients with cardiac cachexia associated with pulmonary hypertension.

METHODS

Patients with primary pulmonary hypertension and cardiac cachexia, as defined by weight loss of more than 6%, underwent diagnostic work up to determine aetiology and severity of pulmonary hypertension according to the World Health Organization and British Cardiac Society criteria. The patients in this study were in WHO functional class III or above, the time since onset of disease was 4.9 (2.1) years, and 70% were immediately centrifuged, plasma separated and stored at −80°C until analysis using established in-house assays. Parametric statistical analysis was done using Sigmasstat v2.0 (SPSS Science, Chicago, Illinois, USA). The PYY, ghrelin, and ADM were compared between BMI, fasting ghrelin, and peak PYY.

RESULTS

There were no differences in age, sex, and BMI between control subjects and patients (table 1). PYY was 60% higher in the patients post-meal at 30 minutes (p = 0.01), 49% higher at 60 minutes (p = 0.02), and 46% higher at 90 minutes (p = 0.04) when compared with the control subjects (fig 1). There was no difference in fasting PYY concentrations (p = 0.42).

Both groups showed the expected pattern of reduction in ghrelin post-meal. The fasting ghrelin concentrations for the patients and control subjects were 704.1 (423) pmol/l and 484.6 (204) pmol/l, respectively (p = 0.13), while the 90

<table>
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<tr>
<th>Table 1 Demographics of patients and control subjects</th>
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<tbody>
<tr>
<td>Cardiac cachexia</td>
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<tr>
<td>Subjects (female) 6/9</td>
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<tr>
<td>Age (years) 38.4 (5.0)</td>
</tr>
<tr>
<td>BMI (kg/m2) 19.8 (1.3)</td>
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<td>Weight loss (kg) 9.0 (0.4)</td>
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</tbody>
</table>

Figure 1 PYY response following standard meal in cardiac cachexia associated with pulmonary hypertension and control subjects (solid circles, patients with cardiac cachexia; open circles, control subjects)
A 28 year old man sustained an isolated stab injury to the left third intercostal space in the mid clavicular line. The weapon was reported to be a kitchen knife 12 cm in length. A transthoracic echocardiogram revealed a pericardial effusion. He was transferred to our department and was taken to theatre for emergency surgery. A median sternotomy was performed. An entry wound was found over the clavicular line. The weapon was taken to theatre for emergency surgery. A transthoracic echocardiogram was performed following weaning from bypass to assess intracardiac structures. This revealed severe regurgitation through the anterior leaflet of the tricuspid valve (panels B and C).

Bypass was re-instituted with bicaval cannulation. The right atrium was opened directly on beating heart and a defect confirmed in the tricuspid leaflet. This was repaired with interrupted 5/0 polypropylene sutures. The patient made an uneventful recovery and was discharged home.

References

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Severe tricuspid regurgitation following a stab to the heart

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