Matsuda index.

QUICKI= 1/[log(fasting insulin) + log(fasting glucose)].33

Matsuda index (MATSUDA) was estimated using the following formula:
10000/square root of [(fasting glucose x fasting insulin) x (mean glucose x mean insulin during OGTT)].34

Hyperandrogenemia

The Free Androgen Index (FAI) was estimated using the following formula:
FAI= [TT (ng/dL)/SHBG (nmol/L)] x 100 (%).35

Statistical analysis

Results are reported as mean values±standard error (S.E.). Statistical significance in the results were accepted at a p-value <0.05. Normal distribution of continuous variables was assessed by applying the non-parametric Kolmogorov–Smirnov test. An independent-sample, two-tailed t-test was used for comparisons between the PCOS women and the control group. The Mann–Whitney U test was performed for variables which were not normally distributed. Correlations between variables were evaluated in the PCOS group only by Pearson’s coefficient except for variables not normally distributed, which were evaluated by Spearman’s coefficient. Relations between categorical variables were estimated by the chi-square test. Multiple regression analysis was performed in the whole population studied in order to evaluate which variable from PCOS presence (1=PCOS, 0=control), BMI, QUICKI predict hemodynamic the parameters studied.

RESULTS

Women with PCOS had a higher grade of hirsutism severity than the control group (P<0.001). The two groups did not differ in smoking habits (P=0.40), positive family history of type 2 diabetes (P=0.17) or cardiovascular disease (P=0.46) (Table 1).

Hormonal and metabolic parameters are shown in Table 2. All subjects studied exhibited normal glucose tolerance. The PCOS group consisted of 11 (40.7%) normal weight, 9 (33.3%) overweight and 7 (26%) obese women. The control group consisted of 15 (55.6%) normal-weight, 6 (22.2%) overweight and 6 (22.2%) obese women. No difference was observed in BMI distribution between the two groups (p=0.52). Women with PCOS had higher levels of TT (P<0.001), Δ4A (P<0.001), LH (P=0.03), higher values of FAI index (P<0.001) and LH-to-FSH ratio (P=0.01) and lower values of QUICKI (P=0.04) and MATSUDA indices (P=0.006).

FMD values were lower in the PCOS group compared to controls (P<0.001), but no difference was observed in NID and in SBP and DBP values (Table 3).

In plethysmography parameters, the only difference was observed in the time to peak reactive hyperemia, which was longer in PCOS women (P=0.02) (Table 3).

No difference was observed in combined IMT values between the studied groups (Table 3).

In women with PCOS, FMD was positively related to QUICKI index (r=0.40, p=0.04) and ne-

<table>
<thead>
<tr>
<th></th>
<th>PCOS [N/total N (%)]</th>
<th>Controls [N/total N (%)]</th>
<th>P for comparison between PCOS and control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ferriman Gallway scale &gt;6</td>
<td>24/27 (88.9)</td>
<td>1/27 (3.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Smokers</td>
<td>9/27 (34.6)</td>
<td>13/27 (48.1)</td>
<td>0.40</td>
</tr>
<tr>
<td>Positive family history for type 2 diabetes</td>
<td>14/26 (53.8)</td>
<td>9/27 (33.3)</td>
<td>0.17</td>
</tr>
<tr>
<td>Positive family history for cardiovascular disease</td>
<td>3/27 (11.1)</td>
<td>5/27 (19.2)</td>
<td>0.46</td>
</tr>
</tbody>
</table>