plasma GSTA1-1 concentration decreased; in 16 women, the plasma GSTA1-1 concentration increased; \( P < 0.0002 \) for the whole group) when compared with estradiol only.

Sex-related differences in the expression of GST isoenzymes in liver were reported in rats [5] and mice [6] but not in humans. In female mice, ovariectomy resulted in increased liver GST activities that could be normalized by replacement of estrogen and progesterone [6]. In humans, men had higher plasma GST-alpha concentrations than women [3, 4]. In women, but not in men, an increase with age was noted [4]. This study shows that estradiol replacement did not decrease the relatively high plasma GSTA1-1 concentrations detected in elderly women. In contrast, the 8–10 days of replacement with estradiol combined with the progestogen NETA resulted in a 20% decrease of the median plasma GSTA1-1 concentration in postmenopausal women, indicating that the relatively high plasma GSTA1-1 concentrations observed in elderly women may be related to the low progestogen concentrations found in these women.

The effect of hormonal replacement therapy on plasma GSTA1-1 concentrations in women reported in this study is on the same order of magnitude as the influence of gender and age on plasma GSTA1-1 concentrations. Although the effects of these variables are rather small, they should be kept in mind when plasma GSTA1-1 concentrations of patients are compared with reference intervals obtained from the general population.

Plasma samples were obtained during a study sponsored by Novo Nordisk Farma BV. This work was supported by grant 28-2801 from the Dutch “Praeventiefonds” (to M.F.C.M.K. and T.P.J.M.).

<p>| Table 1. Plasma GSTA1-1 concentrations in postmenopausal women receiving 17(\beta)-estradiol and NETA in a 28-day (Trisequens(^\circ)) or a 84-day (long-cycle) treatment cycle. |
|---|---|---|
| Plasma GSTA1-1 concentration, µg/L (median and range) | Trisequens | Long cycle | All |</p>
<table>
<thead>
<tr>
<th>n</th>
<th>Baseline</th>
<th>Estradiol only</th>
<th>Estradiol + NETA</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>12</td>
<td>14</td>
<td>26</td>
</tr>
<tr>
<td>Baseline</td>
<td>2.32 (1.72–3.20)</td>
<td>1.94 (1.56–2.46)</td>
<td>2.18 (1.69–2.46)</td>
</tr>
<tr>
<td>Estradiol only</td>
<td>1.99 (1.61–2.57)</td>
<td>2.31 (2.03–2.67)</td>
<td>2.25 (1.96–2.57)</td>
</tr>
<tr>
<td>Estradiol + NETA</td>
<td>1.82 (1.34–2.29)</td>
<td>1.58 (1.30–2.02)</td>
<td>1.81 (1.34–2.29)</td>
</tr>
</tbody>
</table>

The women in the long-cycle group received 2 mg of micronized 17\(\beta\)-estradiol on days 1–78 and 1 mg on days 79–84 and 1 mg of NETA on days 69–78. The women in the Trisequens group received 2 mg of 17\(\beta\)-estradiol on days 1–22 and 1 mg on days 23–28 and 1 mg of NETA on days 13–22. Plasma samples were collected on three different occasions: (a) at the start of the study (baseline); (b) 1–3 days before the last NETA administration (estradiol only, cycle days 66–68 in the long-cycle group, and cycle days 10–12 in the Trisequens group); and (c) on days 8–10 of the last NETA administration (estradiol + NETA; cycle days 76–78 in the long-cycle group and cycle days 20–22 in the Trisequens group).

\( ^a P < 0.03 \) vs estradiol only.

\( ^b P < 0.003 \) vs estradiol only.

\( ^c P < 0.0002 \) vs estradiol only.

Serum Cardiac Troponins I and T in Early Posttraumatic Rhabdomyolysis, Alain Lavoinne* and Guy Hue (Lab. de Biochim. Méd., Centre Hosp. Univ., 1 rue de Germont, 76031 Rouen, France; * author for correspondence: fax (33) 2 32 88 87 80; e-mail Biochimie@chu-rouen.fr)

Troponin T was proposed as an aid in the diagnosis of myocardial infarction in 1989 [1]. However, the first generation of commercially available troponin T assays yielded false-positive results in patients with severe skeletal muscle injury because of an unspecified binding of skeletal muscle troponin T to the wall of the test tube. Indeed, the second detection antibody in the test showed marked cross-reactivity with skeletal muscle troponin T. Another protein of the troponin complex, cardiac troponin I (cTnI), recently became commercially available and was shown to be highly specific for cardiac muscle [2]. cTnI can be used for the diagnosis of myocardial damage in patients with posttraumatic rhabdomyolysis [3]. More recently, a cardiac-specific, second-generation assay for troponin T (cTnT) was developed by using a second completely cardiac-specific antibody [4]. We report here the comparison of the results obtained for cTnI and cTnT in patients with early posttraumatic rhabdomyolysis [creatinine kinase (CK) activity >4000 U/L; upper reference limit (URL), 110 U/L].

Total CK activity was assessed at 30°C according to IFCC recommendations with a Hitachi 704 analyzer (Boehringer Mannheim). The intraassay reproducibility for CK (160 U/L) was 0.9% (n = 10). cTnI (routinely assayed in our laboratory) and cTnT were measured with commercially available immunoassays (Stratus cTnI from Dade and Elecsys cTnT from Boehringer Mannheim). The detection limits for cTnI and cTnT, as assessed by 10 determinations of a sample free of cTnI and cTnT, were 0.10 µg/L and 0.018 µg/L, respectively. Ten determinations of the same serum sample (intraassay reproducibility) showed CVs of 5.4% and 1.3% for cTnI and cTnT, respectively. The mean ± SD concentrations (µg/L) were: 2.79 ± 0.15 and 0.31 ± 0.04, respectively.

References
value was <1.6 µg/L for cTnI. The reported reference value for cTnT was <0.1 µg/L [4]. To verify the URL for cTnT, cTnI and cTnT were measured in 50 patients admitted to our hospital for suspected myocardial infarction. The relationship between cTnI (x) and cTnT (y) was
\[ y = 0.147x - 0.144 \]  
\( \text{r} = 0.900 \). Thus, a theoretical URL of 0.09 µg/L for cTnT was obtained, which is same as that recommended by the manufacturer.

cTnI and cTnT were measured in 15 patients (median age, 59 years; range, 24–90 years; sex, 12 men and 3 women) with early posttraumatic rhabdomyolysis. In blood, free ("ionized") magnesium (Mg\textsuperscript{2+}) is in equilibrium with complexed (protein-bound, and organic or inorganic complexed) species. This equilibrium is influenced by pH and by protein and ligand concentration, both in vivo and in vitro. The phenomenon is well known for calcium; pH influences the serum concentration of free calcium ion (S-cCa\textsuperscript{2+}) through the competition between hydrogen ions and calcium ions toward binding sites of protein. Fogh-Andersen [1] proposed the relationship
\[ dpcCa^{2+}/dpH, \] 
correcting the measured S-cCa\textsuperscript{2+} to the standard pH of 7.4, that, although controversial, is widely used in commercial analyzers [2, 3].

Recently, synthetic neutral carriers for the determination of free magnesium ion concentration (cMg\textsuperscript{2+}) have been developed [4–7], the selectivity of which toward calcium allows the chemometric correction of the free magnesium measurement in the presence of pathophysiological calcium concentrations [8, 9]. Magnesium bound to protein is not measured by the current generation of calcium and magnesium analyzers.