Gender Differences in Regression of Electrocardiographic Left Ventricular Hypertrophy During Antihypertensive Therapy

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Abstract—Although men and women differ in the magnitude of ECG left ventricular hypertrophy, whether gender differences exist in the degree of regression of ECG left ventricular hypertrophy during antihypertensive therapy is unclear. ECG left ventricular hypertrophy defined using gender-adjusted Cornell product and Sokolow-Lyon voltage criteria was assessed serially in 9193 hypertensive patients treated with losartan- or atenolol-based regimens. Changes in ECG left ventricular hypertrophy were measured from baseline to last in-study visit, and above-average regression of hypertrophy was identified by a ≥236-mm · ms reduction in Cornell product or ≥3.5-mm reduction in Sokolow-Lyon voltage. During mean follow-up of 4.8±0.9 years, women had less reduction in Cornell product (−149±823 versus −251±890 mm · ms) and Sokolow-Lyon voltage (−3.0±6.8 versus −4.8±7.7 mm) than men (both P<0.001). After adjusting for baseline ECG left ventricular hypertrophy levels, baseline and change in systolic and diastolic pressures, treatment group, age, and other baseline gender differences, women had significantly less reduction in both Cornell product (adjusted means: −137 versus −276 mm · ms; P<0.001) and Sokolow-Lyon voltage (−3.6 versus −4.1 mm; P=0.005) than men and were 32% less likely to have had greater than the median level of regression of Cornell product left ventricular hypertrophy (95% CI: 24% to 39%; P<0.001) and 15% less likely to have had regression of left ventricular hypertrophy by Sokolow-Lyon criteria (95% CI: 5% to 23%; P=0.003). Thus, women have less regression of ECG left ventricular hypertrophy than men in response to antihypertensive therapy, independent of baseline gender differences in the severity of ECG left ventricular hypertrophy and after taking into account treatment effects and blood pressure changes. (Hypertension. 2008;52:100-106.)

Keys Words: electrocardiography ■ gender ■ hypertension ■ hypertrophy

Men and women have well-established differences in the magnitude of ECG measurements.1–10 Women have shorter QRS durations1–5 and lower QRS voltages,4–10 with consequent parallel gender differences in ECG criteria for left ventricular (LV) hypertrophy (LVH), which incorporate QRS amplitude and duration measurements.5,10 Although gender differences in ECG LVH criteria can be in part attributed to differences between men and women in body size, obesity, and LV mass,5 gender differences in ECG LVH criteria persist even after adjusting for differences in LV mass and body habitus between men and women.5

Reduction of blood pressure (BP) by antihypertensive therapy produces regression of ECG LVH,11–18 and regression of ECG LVH is associated with improved prognosis.11,14,19–24 although less regression of ECG LVH has been reported in subgroups, such as patients with diabetes.18 Several studies have reported gender differences in LV structural and functional adaptations to hemodynamic overload.25–28 Although greater regression of ECG LVH in response to losartan-based therapy in the Losartan Intervention For Endpoint reduction in hypertension (LIFE) Study was demonstrated previously in both men and women,17 whether there were gender differences in the degrees of LVH regression was not examined. Thus, whether there are gender differences in the magnitude of regression of ECG LVH in response to antihypertensive therapy is unclear. Therefore, the present study examined whether men and women have differing regression of ECG LVH by Cornell product and Sokolow-Lyon voltage in response to aggressive antihypertensive therapy in the LIFE Study, taking into account effects of treatment, BP, and other covariates.
Methods

Subjects
The LIFE Trial\(^{17,18,21–24,29}\) enrolled 9193 hypertensive patients with ECG LVH by Cornell voltage-duration product\(^{26}\) and/or Sokolow-Lyon voltage criteria\(^{31}\) in a prospective, double-blind, randomized study to determine whether greater reduction in mortality and morbidity events is associated with the use of losartan as opposed to atenolol. The study was approved by all of the concerned ethics committees. As described previously\(^{17,18,21–24,29}\), eligible patients included men and women aged 55 to 80 years with untreated or treated essential hypertension with mean seated BP in the range from 160 to 200/95 to 115 mm Hg after 1 and 2 weeks on placebo who had not suffered a myocardial infarction or stroke within 6 months and did not require treatment with a \(\beta\)-blocker, angiotensin-converting enzyme inhibitor, or angiotensin receptor antagonist.

Treatment Regimens
Blinded treatment started with 50 mg of losartan or 50 mg of atenolol daily and matching placebo of the other agent, with a target BP of \(\leq 140/90\) mm Hg. During clinic visits at frequent intervals for the first 6 months and at 6-month intervals thereafter, study therapy could be up-titrated by adding 12.5 mg of hydrochlorothiazide, followed by increasing blinded losartan or atenolol to 100 mg daily. If BP was still not controlled, additional open-label upward titration of hydrochlorothiazide and, if necessary, therapy with a calcium channel blocker or other additional medications (excluding angiotensin II type 1, \(\beta\)-blockers, or angiotensin-converting enzyme inhibitors) was added to the double-blind treatment regimen.\(^{29}\)

Electrocardiography
ECGs were obtained at baseline, 6 months, and at 1-year follow-up intervals thereafter until study termination or patient death. QRS duration was measured to the nearest 4 ms, and R-wave and S-wave amplitudes were measured to the nearest 0.05 mV at the LIFE ECG Core Laboratory.\(^{17,18,21–24}\) The products of QRS duration times the Cornell voltage combination (\(R_{aVL} + S_{v5}\), with 6 mm added in women\(^{17,30}\)) \(>2440\) mm \(\cdot\) ms or Sokolow-Lyon voltage (\(SV_1 + RV_5\)) \(>38\) mm\(^{11}\) were used to identify the presence of LVH. A reduction in the Cornell product of \(>236\) mm \(\cdot\) ms or decrease in Sokolow-Lyon voltage of \(>3.5\) mm, the median changes between baseline and last in-study measurement in the overall population,\(^{23}\) were used to define regression of LVH.

Statistical Analyses
Data management and analysis were performed by P.M.O. using SPSS version 12.0. Data are presented as means \(\pm SDs\) for continuous variables and proportions for categorical variables. Differences in mean values between men and women were compared using unpaired \(t\) tests, and comparison of proportions between groups was performed using \(x^2\) tests. Gender differences in mean changes in the Cornell product and Sokolow-Lyon voltage between baseline and the 6-month and each yearly determination were compared using repeated-measures ANCOVA adjusting for baseline Cornell product or Sokolow-Lyon voltage measurements, respectively, which included a time × gender interaction to determine whether there was a significant difference in the time trend of these changes between men and women. Gender differences in mean changes in Cornell product and Sokolow-Lyon voltage between baseline and last in-treatment ECG were further compared after adjusting for gender differences in baseline Cornell product or Sokolow-Lyon voltage, baseline and changes in systolic and diastolic BP, baseline demographic and other clinical differences, and randomized treatment allocation using repeated-measures ANCOVA. The relationship of gender to regression of ECG LVH above or below the median decrease in Cornell product and Sokolow-Lyon voltage between baseline and last in-study ECG was further assessed using univariate and multivariable logistic regression analyses. For all of the tests, 2-tailed \(P < 0.05\) was required for statistical significance.

Results
Clinical and demographic characteristics of female and male patients are shown in Table 1. Compared with men, women were older and less likely to be black; have a history of ischemic heart disease, myocardial infarction, or stroke; and to be current smokers. They also had higher body mass indexes and total and high-density lipoprotein cholesterol levels and lower glucose, creatinine, and uric acid levels but were similar with respect to a history of diabetes, peripheral vascular disease and heart failure, had similar degrees of albuminuria, had higher heart rates, and were equally likely to have been treated with losartan or atenolol. Baseline ECG LVH measurements and baseline BP in relation to gender are also shown in Table 1. Women had higher mean Cornell product, with prespecified gender adjustment, lower mean Sokolow-Lyon voltage and QRS duration at baseline, higher
Changes in systolic and diastolic pressures between baseline and subsequent in-treatment determinations in men and women are compared in Table 2. There were no gender differences in changes in systolic pressure between baseline and any of the subsequent measurement points, including the last in-study determination. Changes in diastolic pressure were similar in men and women over the first 3 years of treatment and were then on-average 1 mm Hg lower in women than in men at year 4, year 5, and at last in-study measurement.

Mean values at baseline, subsequent in-study measurements, and changes between baseline and follow-up measurements for gender-adjusted Cornell product and Sokolow-Lyon voltage are compared between men and women in Table 3. Reductions in ECG LVH by both Cornell product and Sokolow-Lyon voltage criteria were significantly smaller in women than in men, with the smaller reductions becoming apparent after only 6 months of therapy and persisting at each yearly re-evaluation and at last in-study measurement. Gender differences in the reduction of LVH by both Cornell product and Sokolow-Lyon voltage became progressively greater over the course of follow-up, ranging from 45 mm·ms and 0.5 mm at 6 months to 123 mm·ms and 1.7 mm by 5 years of follow-up ($P<0.001$ for differences in the time trend between men and women by the time-gender interaction terms in repeated-measures ANCOVA in Table 2). Gender differences in the reduction in ECG LVH were not dependent on gender differences in absolute magnitude of ECG LVH measures, with women having smaller percentage reductions in Cornell product and Sokolow-Lyon voltage at each assessment and at last in-study determination (−3.2±31.1% versus −6.4±40.7% and −8.9±24.5% versus

### Table 2. Changes in Systolic and Diastolic BPs in Relation to Gender

<table>
<thead>
<tr>
<th>Variables</th>
<th>Men (n=4230)</th>
<th>Women (n=4963)</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Change in systolic BP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline to 6 months</td>
<td>−24±1.6</td>
<td>−25±1.7</td>
<td>0.404</td>
</tr>
<tr>
<td>Baseline to 1 year</td>
<td>−24±1.7</td>
<td>−24±1.7</td>
<td>0.740</td>
</tr>
<tr>
<td>Baseline to 2 years</td>
<td>−26±1.8</td>
<td>−26±1.8</td>
<td>0.705</td>
</tr>
<tr>
<td>Baseline to 3 years</td>
<td>−28±1.8</td>
<td>−27±1.9</td>
<td>0.210</td>
</tr>
<tr>
<td>Baseline to 4 years</td>
<td>−29±1.8</td>
<td>−28±1.8</td>
<td>0.067</td>
</tr>
<tr>
<td>Baseline to 5 years</td>
<td>−30±1.9</td>
<td>−29±1.9</td>
<td>0.182</td>
</tr>
<tr>
<td>Baseline to last in-study measurement</td>
<td>−30±1.9</td>
<td>−29±2.0</td>
<td>0.056</td>
</tr>
<tr>
<td>Change in diastolic BP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline to 6 months</td>
<td>−13±0.9</td>
<td>−13±0.9</td>
<td>0.441</td>
</tr>
<tr>
<td>Baseline to 1 year</td>
<td>−13±0.9</td>
<td>−13±0.9</td>
<td>0.995</td>
</tr>
<tr>
<td>Baseline to 2 years</td>
<td>−14±0.9</td>
<td>−14±0.9</td>
<td>0.263</td>
</tr>
<tr>
<td>Baseline to 3 years</td>
<td>−15±0.9</td>
<td>−15±0.9</td>
<td>0.266</td>
</tr>
<tr>
<td>Baseline to 4 years</td>
<td>−17±0.9</td>
<td>−16±0.9</td>
<td>0.019</td>
</tr>
<tr>
<td>Baseline to 5 years</td>
<td>−18±0.9</td>
<td>−17±0.9</td>
<td>0.001</td>
</tr>
<tr>
<td>Baseline to last in-study measurement</td>
<td>−18±0.9</td>
<td>−17±0.9</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Baseline systolic pressures, and lower baseline diastolic pressures than men. Baseline unadjusted Cornell voltage was significantly lower in women than in men, whereas gender-adjusted Cornell voltage (with 6 mm added to the voltage in women) used for the determination of Cornell product in the LIFE Study$^{17,21-24}$ was significantly greater in women than in men (Table 1).

### Table 3. Cornell Voltage-Duration Product and Sokolow-Lyon Voltage Measurements and Change in Measurements in Relation to Gender

<table>
<thead>
<tr>
<th>Time</th>
<th>Men (n=4230)</th>
<th>Women (n=4963)</th>
<th>$P$ Men vs Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>n</td>
<td>Baseline</td>
<td>Visit</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Cornell product, mm·ms*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Month 6</td>
<td>3693</td>
<td>2700±1031</td>
<td>2554±1062</td>
</tr>
<tr>
<td>Year 1</td>
<td>3808</td>
<td>2700±1028</td>
<td>2498±1068</td>
</tr>
<tr>
<td>Year 2</td>
<td>3629</td>
<td>2699±1027</td>
<td>2433±1061</td>
</tr>
<tr>
<td>Year 3</td>
<td>3456</td>
<td>2692±1014</td>
<td>2410±1071</td>
</tr>
<tr>
<td>Year 4</td>
<td>3325</td>
<td>2695±1018</td>
<td>2414±1090</td>
</tr>
<tr>
<td>Year 5</td>
<td>2395</td>
<td>2719±988</td>
<td>2414±1034</td>
</tr>
<tr>
<td>Last</td>
<td>4078</td>
<td>2714±1047</td>
<td>2463±1149</td>
</tr>
<tr>
<td>Sokolow-Lyon voltage, mm*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Month 6</td>
<td>3706</td>
<td>31.9±10.6</td>
<td>30.1±10.2</td>
</tr>
<tr>
<td>Year 1</td>
<td>3831</td>
<td>32.0±10.6</td>
<td>29.4±10.1</td>
</tr>
<tr>
<td>Year 2</td>
<td>3669</td>
<td>31.9±10.5</td>
<td>28.4±10.0</td>
</tr>
<tr>
<td>Year 3</td>
<td>3486</td>
<td>32.0±10.6</td>
<td>27.9±10.0</td>
</tr>
<tr>
<td>Year 4</td>
<td>3343</td>
<td>32.0±10.6</td>
<td>27.3±9.8</td>
</tr>
<tr>
<td>Year 5</td>
<td>2401</td>
<td>32.1±10.4</td>
<td>27.0±9.8</td>
</tr>
<tr>
<td>Last</td>
<td>4109</td>
<td>32.1±10.7</td>
<td>27.3±10.3</td>
</tr>
</tbody>
</table>

* $P<0.001$ for time-gender interaction comparing time trend in mean changes between men and women.
-13.1 ± 24.5%, respectively; both P < 0.001 for change to last in-study measurement; other data not shown). Of note, these ECG changes mirror parallel analyses in the echocardiographic substudy of LIFE, which reveal significant gender differences in changes in LV mass indexed to height\(^2\), with parallel increasing differences in the mean reduction in indexed LV mass over time between men and women from \(-7.3 ± 6.3\) g/m\(^2\) to \(-12.0 ± 8.9\) g/m\(^2\) after 1 year to \(-11.9 ± 9.1\) g/m\(^2\) after 4 years of treatment (\(P = 0.01\) for time-gender interaction).

Reductions in the prevalence of ECG LVH from study baseline to the final available in-study ECG also paralleled the mean changes in ECG LVH and were less in women than in men for Cornell product LVH (\(-14.7\%\) versus \(-19.0\%; \(P < 0.001\)) and Sokolow-Lyon voltage LVH (\(-6.1\%\) versus \(-14.9\%; \(P < 0.001\)). In addition, univariate logistic regression analyses to assess the relationship of gender to reduction of ECG LVH indices more than the median value in the population between baseline and last in-study ECG (Table 4) demonstrated that female gender remained associated with a 32% lower likelihood of above-average regression of Cornell product LVH and with a 15% lower likelihood of above-average regression of LVH by Sokolow-Lyon voltage criteria after taking these other factors into account. Of note, alternatively defining regression of LVH to have occurred when Cornell product or Sokolow-Lyon voltage decreased to below their partition values of 2440 mm ∙ ms and 38 mm, respectively did not change the degree of observed gender differences. Female gender had similar associations with regression of Cornell product LVH (hazard ratio: 0.69; 95% CI: 0.61 to 0.79) and regression of Sokolow-Lyon voltage (hazard ratio: 0.83; 95% CI: 0.69 to 0.98) as when LVH regression is defined by the median decreases in these criteria.

Because gender differences in changes in Cornell product and Sokolow-Lyon voltage could be related to baseline levels of Cornell product and Sokolow-Lyon voltage and could be affected by gender differences in baseline and changes in systolic and diastolic pressure and demographic and clinical variables, and the known treatment differences in regression of ECG LVH,\(^{17}\) the relationships of changes in Cornell product and Sokolow-Lyon voltage between baseline and last in-study determination to gender were further assessed after adjusting for these differences (Figure and Table 4). Adjusted mean reductions in both Cornell product and Sokolow-Lyon voltage remained significantly smaller in women than in men after taking into account treatment assignment and gender differences in covariates that might impact LVH regression (Figure). Importantly, these gender differences persisted and were similar in both obese (body mass index: > 30 kg/m\(^2\)) and nonobese patients and in both treatment groups. Multivariable logistic regression analyses examining the relationship of gender to regression of ECG LVH more than median values (Table 4) demonstrated that female gender remained associated with a 32% lower likelihood of above-average regression of Cornell product LVH and with a 15% lower likelihood of above-average regression of LVH by Sokolow-Lyon voltage criteria after taking these other factors into account. Of note, alternatively defining regression of LVH to have occurred when Cornell product or Sokolow-Lyon voltage decreased to below their partition values of 2440 mm ∙ ms and 38 mm, respectively did not change the degree of observed gender differences. Female gender had similar associations with regression of Cornell product LVH (hazard ratio: 0.69; 95% CI: 0.61 to 0.79) and regression of Sokolow-Lyon voltage (hazard ratio: 0.83; 95% CI: 0.69 to 0.98) as when LVH regression is defined by the median decreases in these criteria.

**Figure.** Gender differences in mean change in Cornell product and Sokolow-Lyon voltage between baseline and last in-study measurement, adjusted for age, race, treatment group allocation, baseline body mass index, baseline diabetes, history of ischemic heart disease, myocardial infarction and stroke, smoking history, baseline high-density lipoprotein and total cholesterol, creatinine, hemoglobin, glucose and uric acid, baseline diastolic and systolic BPs, change in diastolic and systolic BPs between baseline and last in-study measurement, and either baseline Cornell product or baseline Sokolow-Lyon voltage. (Values are reported with SEs of the adjusted mean measurements.)
Discussion
These observations in the LIFE Study cohort demonstrate that women have less regression of ECG LVH than men by both Cornell product and Sokolow-Lyon voltage in the setting of similarly aggressive BP lowering. Lesser reductions in Cornell product and Sokolow-Lyon voltage persist after adjusting for treatment group, baseline gender differences in age, race, systolic and diastolic BP, severity of ECG LVH, and for other covariates; as well as for the slightly smaller reductions in systolic and diastolic pressures in women during the study. These findings appear to parallel other gender differences in adaptation to hypertension and its treatment.\(^{25-28}\) including a higher prevalence of LVH among women than men in hypertensive populations that included previously treated patients.\(^{32,33}\)

Previous studies have found significant gender differences in the magnitude of ECG LVH criteria, with women having lower QRS amplitudes, shorter QRS durations, and, as a consequence, lower levels of ECG LVH criteria based on QRS duration and amplitudes.\(^{1-10}\) differences that are incompletely explained by gender differences in LV mass, body size, and obesity.\(^{5}\) Regression of ECG LVH in response to antihypertensive therapy has been demonstrated in a number of populations, and we have reported previously that losartan-based therapy was associated with greater regression of Cornell product and Sokolow-Lyon voltage LVH than atenolol-based therapy in both men and women in the LIFE Study.\(^{17}\) To our knowledge, this is the first study to demonstrate lesser regression of ECG LVH in women than in men. Importantly, the smaller reductions in ECG LVH in women became apparent early in the course of therapy, became greater throughout the study (Table 2), and were independent of other possible confounders and gender differences, including the slightly smaller reductions in systolic and diastolic pressure in women (Table 2). As a result, women had 15% to 32% lower likelihoods of having above-average regression of ECG LVH indices between baseline and last in-study measurement in multivariable analyses (Table 4). In addition, gender differences in the change in echocardiographic LV mass appear to directly parallel these findings for ECG LVH, suggesting that lesser regression of LVH in women in response to antihypertensive treatment is not dependent on the methodology used to assess LVH.

Several mechanisms may play a role in these gender differences in the regression of LVH. First, lesser regression of ECG LVH in women despite similar reductions in systolic and diastolic pressures may reflect gender differences in central arterial pressure waveforms\(^{34}\) that impact on the hypertrophy process.\(^{34,35}\) Adult women have significantly higher systolic pressure augmentation indexes\(^{34}\) characterized by an increased contribution of a secondary, late systolic peak to central aortic pressure. Given that late-peaking systolic pressure is associated with greater LVH,\(^{35}\) even with similar systolic BP,\(^{34}\) and in light of findings that the ability of pharmacological agents to limit LVH in a rat model of hypertension have been linked to their effect on pulsatile vascular afterload,\(^{36}\) one can speculate that the lesser regression of ECG LVH observed in the current study may, in part, reflect a greater central aortic pressure load on the LV in women despite similar decreases in brachial systolic pressure. Of note, additional adjustment for gender differences in height, which may in part account for differences in late systolic central aortic pressure between men and women, did not alter the gender differences in changes in ECG LVH observed in the current study. Further investigation of gender differences in LVH regression in relation to augmentation index will be necessary to elucidate the degree to which gender differences in vascular afterload account for differences in LVH regression between men and women.

Gender differences in LVH regression are further supported by observed gender differences in LV remodeling in response to hemodynamic loads.\(^{26,37}\) Luchner et al\(^{26}\) demonstrated more rapid induction of LVH in men than in women in response to hemodynamic overload, with similar degrees of elevation in systolic pressure associated with more severe LVH in men. In addition, lower message levels of sarco/endooplasmic reticulum Ca\(^{2+}\)-ATPase-2 in male compared with female rats with similar degrees of LVH in response to pressure overload\(^{37}\) suggest that the gain of mechanotransduction of systolic load may be higher in male compared with female hearts.

Several potential limitations of the present study should be noted. Because patients in the LIFE Study were selected based on elevated Cornell product and Sokolow-Lyon voltage and moderate- to-severe hypertension,\(^{29}\) the present findings cannot necessarily be extrapolated to less-selected patients with milder hypertension and less evidence of end-organ damage at initiation of treatment. In addition, because patients were selected on the basis of elevated ECG LVH measures, some degree of the decreases in these values, particularly over the initial 6 months, could in part reflect regression to the mean.\(^{38}\) However, changes because of regression to the mean would not interfere with interpretation of the observed gender differences, because these effects should be randomly distributed across the population independent of gender. Third, it is likely that ambulatory BP measurements would provide a more precise measure of the impact of treatment on BP than the office-based determinations used in the current study. Finally, the use of gender-specific Cornell product criteria appeared to select for somewhat greater ECG LVH by gender-adjusted Cornell product criteria in women, whereas the use of nongender-specific Sokolow-Lyon voltage criteria had the opposite impact, with lesser severity ECG LVH by this criterion in women. However, it is unlikely that these selection criteria impacted on the current findings given that women had lesser regression of ECG LVH by both criteria and that the differences in LVH regression between men and women persisted even after adjusting for baseline gender differences in the severity of ECG LVH.

Perspectives
Thus, LIFE demonstrated significantly less regression of ECG LVH by Sokolow-Lyon voltage and Cornell product criteria in women than in men in the presence of comparable BP lowering by losartan- or atenolol-based therapy, with adjustment for other factors that could potentially impact on LVH regression. In light of the absence of a significant
interaction between regression of ECG LVH by Cornell product and Sokolow-Lyon voltage criteria with gender in Cox models demonstrating lower CV morbidity and mortality in LIFE in the setting of LVH regression.\textsuperscript{22,23} these findings suggest that women may derive less prognostic benefit because of their lesser magnitude and lower likelihood of significant LVH regression during antihypertensive therapy. Further examination of gender-specific changes in ECG LVH during treatment will be required to more fully understand this important issue.

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References


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