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Assessment of serum sodium to urinary sodium divided by (serum potassium)² to urinary potassium as a screening tool for primary aldosteronism

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Abstract

Background: The SUSPPUP ratio (serum sodium/urinary sodium)/(serum potassium²/urinary potassium) has been proposed as a marker to screen for primary aldosteronism (PA). The original study found an area under the receiver operating characteristic (ROC) curve of 0.90 to detect PA; the sensitivity was 89% and specificity 86% for a ratio over 5.3 l/mmol.

Materials and methods: Patients attending a hypertension unit between 2001 and 2006 and for who renin and aldosterone measurements and concomitant serum and urinary biochemistry data were available were included if diagnosed with PA (n=449) or essential hypertension (n=2209). We compared the diagnostic value of the SUSPPUP ratio and of serum potassium in the whole population, in patients without interfering drugs and in patients with lateralized PA.

Results: The area under the ROC curve was significantly worse for the SUSPPUP ratio than for serum potassium in all groups: 0.72 vs. 0.76 in the whole population; 0.73 vs. 0.78 without interfering drugs; 0.76 vs. 0.82 for patients with lateralized PA.

In the whole population, sensitivity was 71% for a SUPPUP ratio ≥ 5.3 l/mmol and serum potassium < 3.7 mmol/l, but specificity of the SUSPPUP ratio was significantly worse (61% vs. 69%). Using low serum potassium and/or high SUSPPUP ratio increased the sensitivity to 87% but decreased the specificity to 47%.

Conclusions: The SUSPPUP ratio was outperformed by serum potassium as a screening tool for PA in this large validation sample. Its value as an adjunct to serum potassium is questionable because of the low specificity of their combination.

Keywords
Hypertension; primary aldosteronism; potassium, blood; potassium, urine; diagnosis; sensitivity and specificity
Introduction
The frequency of primary aldosteronism (PA) is at most 10% among unselected hypertensive patients, and probably less [1]. The plasma aldosterone to renin ratio (ARR) is the recommended screening test for PA [2]. However, widespread hormonal screening would result in many false positives and subsequent unnecessary expensive or even harmful diagnostic procedures [3, 4]. Guidelines therefore still advocate screening for PA only in cases of low serum potassium, resistant hypertension or adrenal mass [2, 5-7]. Low serum potassium has for a long time been considered to be a good indicator of PA, but is currently reported in as few as 10 to 40% of patients [8]. PA with normal serum potassium is most likely to be due to bilateral adrenal hyperplasia [3, 9] but lateralized PA can also present with normal serum potassium in a significant proportion of cases [10].

The SUSPPUP ratio (serum sodium/urinary sodium)/(serum potassium²/urinary potassium) has been proposed as a cheap tool to detect additional patients who would qualify for hormonal screening. The basis for this suggestion was a pathophysiological rationale: excess secretion of aldosterone promotes potassium secretion, and sodium and water retention. The SUSPPUP ratio was designed to detect relative high serum and low urinary sodium concentrations, along with relatively low serum and high urinary potassium concentrations. The original study found an area under the receiving operator characteristic (ROC) curve of 0.90 to detect PA; the sensitivity was 89% and specificity 86% for a SUSPPUP ratio over 5.3 l/mmol [11]. These results were supported by a second small study, where the area under the ROC curve was 0.87, the sensitivity was 85% and the specificity 82% for a SUSPPUP ratio over 5.95 l/mmol [12]. We undertook a retrospective study to validate this ratio in a large sample of patients with PA or essential hypertension (EH).

Patients and methods
Patients and data
Consecutive patients who underwent renin and aldosterone measurements in our hypertension unit between January 1, 2001, and December 31, 2006 were retrospectively identified and included if finally diagnosed with PA or EH [13]. Patients for who urine biochemistry data were unavailable and PA patients with severely impaired renal function (estimated glomerular filtration rate < 30 ml/min/1.73m²) were excluded.

As a general rule, renin and aldosterone measurements were performed in patients fulfilling at least one of the following criteria: hypertension onset at an age below 40 years old; resistant (BP > 140/90 mmHg on three or more antihypertensive drugs including a diuretic) or severe (BP > 180/110 mmHg) hypertension; or hypertension associated with serum potassium < 3.9 mmol/l (or < 3.6 mmol/l if on thiazide or loop diuretic) [5].

Clinical data (age, sex, blood pressure, prescribed drug classes) and biological data (serum and urinary sodium and potassium, serum creatinine, plasma renin, plasma and urinary aldosterone) were collected from the first in-patient visit for a diagnostic workup for all patients, irrespective of their final diagnosis. Clinical data from all patients were extracted from the electronic health record database, where they had been entered prospectively as part of routine care [14]. Laboratory results were extracted from the database of the biochemistry department.

Test methods and diagnostic criteria
Blood samples were drawn after washout of interfering drugs whenever possible (for at least two weeks for diuretics, beta-blockers, ACE inhibitors and angiotensin antagonists and at least six weeks for spironolactone and aliskiren). Potassium supplements were prescribed if required to avoid severe hypokalemia that could affect hormonal measurements. A venous catheter was inserted on patients’ admission and free-flowing blood was drawn after 1 hour of lying or seating rest. In patients with PA or EH for whom aldosterone measurements in the lying and sitting positions were available, we observed that both values were similar. Therefore, we used aldosterone and renin levels in the lying position when available and in the sitting position otherwise. Urinary sodium and potassium were determined from a 24-hour urine collection.

Plasma and urinary aldosterone concentrations were determined by radioimmunoassay (Coat-A-Count®, Siemens Medical Solutions Diagnostics); 24-hour urinary excretion of aldosterone was measured as the sum of free aldosterone and aldosterone from the hydrolysis of aldosterone 18-glucuronide at pH 1.0. Plasma active renin was determined by chemiluminescent immunoassay (LIAISON®, Diasorin). Serum and urinary potassium concentrations were determined by indirect potentiometry (Unicel DxC 800® system, Beckmann Coulter). All of the other biochemical variables were assayed in plasma or serum by standard methods.

PA was diagnosed if the ARR obtained in standardized conditions was > 64 pmol/mIU (107 pmol/ng) on two occasions, and if the plasma aldosterone concentration was > 550 pmol/l (20 ng/dl) in the standing or sitting position, or > 500 pmol/l (18 ng/dl) in the supine position, or urinary aldosterone excretion was > 63 nmol/d (23 µg/d). To calculate the ARR, active renin concentrations < 5 mIU/l (3 ng/l) were set at 5 mIU/l to avoid ARR overestimation when active renin concentrations were undetectable or very low.

Patients with PA were operated on the basis of a typical solitary adenoma on CT scan or of the results of adrenal venous sampling (AVS). Patients were classified as having definitely lateralized PA (i) if the aldosterone:cortisol ratio was at least five times higher in one adrenal vein than the other, regardless of postoperative outcome, or (ii) if they did not undergo AVS but were cured by unilateral adrenalectomy (hormonal cure when postoperative values were available and weaning from all antihypertensive drugs with resolution of hypertension and hypokalemia in other cases).

Secondary hypertension was ruled out by a standardised work-up to allow the diagnosis of EH. Renovascular hypertension was defined as renal artery stenosis exceeding a 60% reduction in luminal diameter (due to atherosclerosis of fibromuscular dysplasia) on contrast CT scan. Hypertension was considered secondary to chronic kidney disease in patients with pre-existing parenchymal renal disease, indicated by an estimated glomerular filtration rate < 60 ml/min/1.73m² or proteinuria. Cushing syndrome was excluded by urinary free cortisol < 250 nmol/d and pheochromocytoma by a urinary metanephrines:creatinine ratio < 0.354 [15].

Statistical analysis
Our first objective was to compare the areas under the ROC curve of the SUSPPUP ratio and of serum potassium for the diagnosis of PA. We ascertained that our sample size was sufficient for statistically and clinically meaningful conclusions. Given our sample size, we could expect standard errors of at most 0.015 for our areas under the ROC curves. This allowed a power over 0.99 to show a difference of 0.1 or more between areas under the ROC curves of the SUSPPUP ratio and serum potassium (with an α–level at 0.05).
The secondary objective was to compare the diagnostic accuracy (sensitivity and specificity) of these tests. For these computations, the threshold was set at 5.3 l/mmol for the SUSPPUP ratio, as suggested by the original publication [11]. We also evaluated the SUSPPUP ratio with the threshold at 5.95 l/mmol, reported as optimal in another study [12], and at the threshold with the best Youden index (sensitivity + specificity – 1) in our population. French guidelines recommend looking for PA in cases with serum potassium < 3.9 mmol/l without thiazide or loop diuretic, or < 3.6 mmol/l under such diuretics [5]. The comparison with the SUSPPUP ratio required the use of a single threshold value and we chose the intermediate value of 3.7 mmol/l.

We compared the diagnostic values of the SUSPPUP ratio and of serum potassium in the whole population, in patients without interfering drugs and in patients with lateralized PA. These two subgroups were chosen because patients were weaned from interfering drugs in the original study [11], and because missing patients with bilateral PA is of less importance than missing patients with lateralized PA, who can benefit from surgery. We also evaluated the added value of the SUSPPUP ratio to identify PA patients with normal serum potassium.

Descriptive statistics are reported as medians (interquartile range) or numbers (percentage), as appropriate, and differences were evaluated by the Mann-Whitney and the Chi-square tests, respectively. Areas under the ROC curve for the diagnosis of PA and their 95% confidence intervals were computed for the SUSPPUP ratio and serum potassium, and compared as proposed by Hanley and McNeil [16]. The sensitivity and specificity of tests were compared with the McNemar Chi-square. All statistical analyses were performed with Stata 9.2 (StataCorp, Texas).

The reporting of this study complies with the STARD statement (STAndards for the Reporting of Diagnostic accuracy studies) [17, 18].

Results
During the study period, 459 patients were diagnosed with PA and 2275 patients had a complete workup excluding secondary hypertension. Urinary biochemistry or serum sodium data was missing for 10 patients with PA and 87 patients with EH. Five other PA patients had an estimated GFR < 30 ml/min/1.73 m². We therefore included 444 patients with PA and 2188 patients with EH in the study.

Their clinical, biochemical and hormonal characteristics are given in Table 1. Patients with PA had significantly lower plasma potassium concentrations and higher SUSPPUP ratios than patients with EH, but the distributions overlapped.

PA was lateralized in 115 patients; these patients differed from those for other PA patients for the following biochemical and hormonal variables: serum potassium (3.3 [interquartile range: 3.0, 3.6] mmol/l in patients with lateralized PA vs. 3.4 [3.2, 3.7] mmol/l in other PA patients, p<0.001), SUSPPUP ratio (8.5 [5.6, 13.1] vs. 6.8 [4.9, 11.0] l/mmol, p=0.01), serum aldosterone (644 [441, 1050] vs. 538 [403, 739] pmol/l, p=0.002), serum renin (2.1 [1.4, 4.4] vs. 3.2 [1.9, 5.2] mIU/l, p=0.005), and ARR (122 [85, 193] vs. 95 [72, 133] pmol/mIU, p<0.001).

The area under the ROC curve and specificity of serum potassium (threshold at 3.7 mmol/l) were significantly better than those of the SUSPPUP ratio (threshold at 5.3 l/mmol) in the
whole population and in both prespecified subgroups (Table 2). In the whole population, the best Youden index was found for a SUSPPUP ratio at 5.1 l/mmol, with a sensitivity of 76\% [95\% CI: 71, 79] and a specificity of 57\% [55, 59]; at this threshold, the sensitivity of the SUSPPUP ratio was similar to that of serum potassium with a threshold at 3.7 mmol/l \(p = 0.15\), but the specificity was significantly worse \(p < 0.001\). With a threshold at 5.95 l/mmol, the SUSPPUP ratio had a sensitivity of 63\% [58, 68] and a specificity of 69\% [67, 71] in the whole population; this specificity was similar to that of serum potassium with a threshold at 3.7 mmol/l \(p = 0.68\), but the sensitivity was significantly worse \(p = 0.001\).

A SUSPPUP ratio \(\geq 5.3\) l/mmol was observed in about half of the patients with false negative serum potassium results (Table 3.A). Considering that patients were screen-positive if they had a serum potassium < 3.7 mmol/l or a serum potassium \(\geq 3.7\) mmol/l but a SUSPPUP ratio \(\geq 5.3\) l/mmol significantly increased the overall sensitivity to 87\% [95\% CI: 84, 90] but also significantly decreased the specificity to 47\% [45, 49].

**Discussion**

**Main results**

The SUSPPUP ratio was less accurate than serum potassium to distinguish PA or lateralized PA from EH in this population, owing to a worse specificity for a similar sensitivity or a worse sensitivity for a similar specificity, depending on the choice of the threshold values. Results did not differ significantly in patients who were assessed after washout of interfering drugs. The distribution of serum potassium and the SUSPPUP ratio suggested there may potentially be value in combining these two tests. However, although using the SUSPPUP ratio for patients with normal serum potassium significantly increased sensitivity, it also decreased specificity compared to using serum potassium values alone. As a result, many additional patients would be investigated for PA but only a few of these would be confirmed.

**Comparison with previous studies**

The SUSPPUP performed worse in our population than in the original study where the area under the ROC curve was 0.90 [11], or than in the subsequent study where the area under the ROC curve was 0.87 [12]. Several differences in protocol and in population between the original study and ours must be outlined.

We determined the SUSPPUP ratio with urinary sodium and potassium concentrations measured from 24-hour urine, whereas a sample of the second miction urine was used in the original study [11]. Although 24-hour urine collections are usually considered as the reference for sodium and potassium measurements, second miction samples may give slightly different and more useful information for the SUSPPUP ratio. However, the fact that Balas et al., who also used 24-h urine, found a diagnostic value close to the original study argues against this hypothesis [12].

Our control patients with EH were selected patients referred to a hypertension unit, in whom hormonal screening was indicated according to guidelines. This may have decreased the specificity of the SUSPPUP ratio, but would then also have decreased the specificity of serum potassium. Moreover, our patients with PA were not selected and much more numerous than in the original study. We therefore consider our estimate of the sensitivity of the SUSPPUP ratio to be more representative and precise than that of the original study. Not all patients could be assessed without interfering drugs, but results were similar when only patients without interfering drugs were considered.
The diagnostic value of the SUSPPUP ratio may have been overestimated in the original study, because of two potential levels of overfitting. First, the best threshold observed in the original study may differ from the true value due to the inaccuracies of estimates in a small sample. Second, the original study did not only evaluate the SUSPPUP ratio, but also the SUSPUP ratio (where urinary potassium is not squared). When several prediction models are evaluated in a small data sample, there is again a risk of selecting the model that fits better with the sample rather than the truly best one.

**Study limits**
A recent guideline advocates the use of suppression tests to confirm PA in patients with a high ARR [2], but the evidence to support this recommendation is weak. We rather require the demonstration of high levels of plasma or urinary aldosterone to confirm PA [19]. Nonetheless, our findings hold true in the large subgroup of patients with lateralized aldosterone hypersecretion, in whom the diagnosis of PA can be regarded as well established.

The distribution of serum potassium in PA patients varies widely among series, as illustrated by differing frequencies of hypokalemia. In a large study with very stringent criteria for PA and a very careful blood sampling procedure (no tourniquet, no fist clenching), only 30% of PA patients had a serum potassium ≤ 3.5 mmol/l [9]. By contrast, 69% of PA patients had a serum potassium ≤ 3.5 mmol/l in the original study evaluating the SUSPPUP ratio [11] and 54% in the current series. Although the observed sensitivity of serum potassium and that of the SUSPPUP ratio depend on the distribution of serum potassium in the study population, this does not necessarily change the result of their comparison if both tests are affected similarly. Our results in patients with lateralized PA support this hypothesis: serum potassium and the SUSPPUP ratio had a higher sensitivity in this more severe subgroup than in the overall population, but the changes were similar for both tests and did not affect their comparison.

**Implications for clinical practice and future research**
Most of the hypertensive patients enrolled in this or in the original study were already being treated for hypertension. As numerous antihypertensive treatments interact with the renin – angiotensin – aldosterone system and influence serum potassium and sodium levels, attempts were made in both studies to withdraw interfering drugs before biochemical and hormonal investigations. Once this effort has been made, it seems sensible to determine the aldosterone:renin ratio regardless of any prior test like the SUSPPUP ratio.

A more relevant issue is whether the SUSPPUP ratio could identify patients with newly diagnosed hypertension for who hormonal screening could possibly be performed beyond current indications (onset before 30 years old, severe hypertension from the onset, low serum potassium at diagnosis). Neither the first study nor ours provide a definite answer to this question, but the pathophysiological reasoning proposed to support the SUSPPUP ratio is questionable. PA is a chronic disease and patients are assessed in a steady-state. Assuming no gastro-intestinal loss, sodium and potassium in the urine reflect no more than sodium and potassium consumption. Patients with PA and patients with EH are therefore likely to have comparable urinary biochemistry if they eat comparable diets. By contrast, serum sodium is consistently higher in patients with PA than in patients with EH; consequently, it may indeed be useful to consider serum sodium as well as serum potassium to increase the diagnostic yield. However, the SUSPPUP ratio is not very sensitive to changes in serum sodium: a clinically significant increase from 137 to 143 mmol/l, for example, results only in a 4% increase of the SUSPPUP ratio. It may be more profitable to investigate diagnostic scores or
rules using both biochemical data and clinical characteristics to better identify patients who could benefit from hormonal screening for PA.

**Acknowledgements**
The authors have no conflict of interest to disclose regarding this study.
References


Table 1. Clinical and biological characteristics of patients with primary aldosteronism or essential hypertension, as medians [interquartile range] or numbers (percent)

<table>
<thead>
<tr>
<th></th>
<th>Primary aldosteronism</th>
<th>Essential hypertension</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>51 [44, 58]</td>
<td>53 [43, 61]</td>
<td>0.05</td>
</tr>
<tr>
<td>Male sex</td>
<td>295 (66%)</td>
<td>1162 (53%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Non-interfering treatment</td>
<td>321 (76%) *</td>
<td>1445 (66%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>148 [134, 166]</td>
<td>148 [133, 166]</td>
<td>0.84</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>88 [79, 96]</td>
<td>86 [78, 96]</td>
<td>0.32</td>
</tr>
<tr>
<td>GFR (ml/min/1.73m²)</td>
<td>86 [71, 100]</td>
<td>86 [73, 99]</td>
<td>0.56</td>
</tr>
<tr>
<td>Serum potassium (mmol/l)</td>
<td>3.4 [3.1, 3.7]</td>
<td>3.8 [3.6, 4.0]</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Serum sodium (mmol/l)</td>
<td>141 [140, 143]</td>
<td>140 [139, 142]</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Urinary potassium (mmol/l)</td>
<td>43 [32, 57]</td>
<td>39 [29, 54]</td>
<td>0.002</td>
</tr>
<tr>
<td>Urinary sodium (mmol/l)</td>
<td>72 [51, 94]</td>
<td>83 [60, 114]</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>SUSPPUP ratio (l/mmol)</td>
<td>7.1 [5.1, 11.4]</td>
<td>4.6 [3.4, 6.5]</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Plasma aldosterone (pmol/l)</td>
<td>556 [407, 779]</td>
<td>236 [157, 330]</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Plasma renin (mUI/l)</td>
<td>3 [1.6, 4.9]</td>
<td>9.1 [4.3, 17.2]</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>ARR (pmol/mUI)</td>
<td>101 [73, 143]</td>
<td>23 [13, 39]</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

* Data on treatment was unavailable for 19 PA patients

BP: Blood Pressure; GFR: Glomerular Filtration Rate; SUSPPUP ratio: (serum sodium/urinary sodium)/(serum potassium²/urinary potassium); ARR: Aldosterone to Renin Ratio
Table 2. Accuracy of the SUSPPUP ratio and of serum potassium to diagnose primary aldosteronism

<table>
<thead>
<tr>
<th></th>
<th>High SUSPPUP ratio</th>
<th>Low serum potassium</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Whole population</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AUC [95% CI]</td>
<td>0.72 [0.69, 0.75]</td>
<td>0.76 [0.74, 0.79]</td>
<td>0.003</td>
</tr>
<tr>
<td>Sensitivity [95% CI] *</td>
<td>71% [67, 76]</td>
<td>72% [67, 76]</td>
<td>0.87</td>
</tr>
<tr>
<td>Specificity [95% CI] *</td>
<td>61% [59, 63]</td>
<td>69% [67, 71]</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Neutral treatment</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AUC [95% CI]</td>
<td>0.73 [0.70, 0.76]</td>
<td>0.78 [0.75, 0.81]</td>
<td>0.01</td>
</tr>
<tr>
<td>Sensitivity [95% CI] *</td>
<td>74% [69, 79]</td>
<td>74% [69, 78]</td>
<td>0.92</td>
</tr>
<tr>
<td>Specificity [95% CI] *</td>
<td>60% [57, 62]</td>
<td>70% [67, 72]</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Lateralized PA</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AUC [95% CI]</td>
<td>0.76 [0.72, 0.81]</td>
<td>0.82 [0.77, 0.86]</td>
<td>0.04</td>
</tr>
<tr>
<td>Sensitivity [95% CI] *</td>
<td>79% [70, 86]</td>
<td>80% [72, 87]</td>
<td>0.72</td>
</tr>
<tr>
<td>Specificity [95% CI] *</td>
<td>61% [59, 63]</td>
<td>69% [67, 71]</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

* threshold 5.3 l/mmol for the SUSPPUP ratio and 3.7 mmol/l for serum potassium

SUSPPUP ratio: (serum sodium/urinary sodium)/(serum potassium²/urinary potassium); AUC: area under the ROC curve; CI: confidence interval; PA: primary aldosteronism
Table 3. SUSPPUP ratio and serum potassium in the overall study population

<table>
<thead>
<tr>
<th></th>
<th>SUSPPUP ratio &lt; 5.3 l/mmol</th>
<th>SUSPPUP ratio ≥ 5.3 l/mmol</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. Primary aldosteronism</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum potassium &lt; 3.7 mmol/l</td>
<td>56 (13%)</td>
<td>69 (15%)</td>
<td>125 (28%)</td>
</tr>
<tr>
<td>Serum potassium ≥ 3.7 mmol/l</td>
<td>71 (16%)</td>
<td>248 (56%)</td>
<td>319 (72%)</td>
</tr>
<tr>
<td>Total</td>
<td>127 (29%)</td>
<td>317 (71%)</td>
<td>444 (100%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>B. Essential hypertension</strong></th>
<th>SUSPPUP ratio &lt; 5.3 l/mmol</th>
<th>SUSPPUP ratio ≥ 5.3 l/mmol</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum potassium &lt; 3.7 mmol/l</td>
<td>1,027 (47%)</td>
<td>473 (22%)</td>
<td>1,500 (69%)</td>
</tr>
<tr>
<td>Serum potassium ≥ 3.7 mmol/l</td>
<td>299 (14%)</td>
<td>389 (17%)</td>
<td>688 (31%)</td>
</tr>
<tr>
<td>Total</td>
<td>1,326 (61%)</td>
<td>862 (39%)</td>
<td>2,188 (100%)</td>
</tr>
</tbody>
</table>