Are low aminoglycoside doses appropriate when used for perioperative prophylaxis in urology?

To the Editor: Peri-operative antibiotic prophylaxis before urological surgery reduces the risk of postoperative urinary tract infection.1 When used in this setting, aminoglycosides are an appropriate choice to prevent infections caused by enteric Gram-negative bacilli. However, prophylactic doses employed are often low, and we feel it appropriate to raise a few points regarding aminoglycoside dosage regimens in this context.

The role of higher aminoglycoside doses once daily has gained widespread acceptance. Aminoglycosides demonstrate concentration-dependent killing of bacteria — the higher peak level, the more rapid the bactericidal activity. Additionally, aminoglycosides exhibit a post-antibiotic effect: bacterial growth is inhibited even when serum antibiotic levels fall to below the minimum inhibitory concentration of that organism. A single daily dose reduces the risk of toxicity since lower trough levels are maintained for longer. Therefore, once-daily doses of aminoglycosides maintain the efficacy of the antibiotic, while reducing the potential risk of side-effects.1,2

When using aminoglycosides perioperatively for urological prophylaxis, it would seem logical to use a once-off standard dose (such as 5 - 7 mg/kg/day for gentamicin). Currently, some regimens utilise gentamicin doses as low as 80 mg3 (equating to about 1 mg/kg), possibly because of concerns about aminoglycoside toxicity. Although aminoglycosides are concentrated in the urine, serum levels attained using these low doses would almost certainly be subtherapeutic. Therefore, if there are concerns about toxicity, an alternative agent such as a quinolone could be employed.4 However, in otherwise healthy individuals we cannot see the rationale behind using low doses of aminoglycosides as perioperative prophylaxis in urological surgery.

A Whitelaw
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The role of nutrient intake in the aetiology of diabetes mellitus

To the Editor: Diabetes mellitus (DM) is a worldwide public health problem, made more acute in Africa by low socio-economic standards. Evidence is also accumulating that dietary factors such as compromised micronutrient or antioxidant status may be aetiological or predisposing factors in DM.1 The aim of this study was to determine the possible role of nutrient intake in the aetiology of DM.

One hundred and three subjects (72 female and 31 male) who showed impaired glucose tolerance (IGT) according to the 1985 World Health Organisation (WHO) criteria2 in 1991 were reinvestigated after 4 years. Seventy-eight subjects (55 female and 23 male) took part in the follow-up study. Of the remaining 25 subjects, 12 died, 4 left the area, 6 chose not to participate and 3 were too ill to participate in the study. Body mass index (BMI) and body fat percentage were determined using bioimpedance (Bodystat) in both the baseline and follow-up studies. Two registered dieticians assessed dietary intake by means of a quantitative food frequency questionnaire. Food models and household measures were used as visual aids to quantify food intake. Dietary data were analysed for nutrients using a mainframe computer program based on the Research Institute for Nutritional Diseases (RIND) Food Composition Tables.3 Nutrient intake was calculated as a percentage of the US recommended dietary allowances (RDA).4 IGT was based on a 120-minute stimulated plasma glucose concentration of 7.8 - 11.1 mmol/l after 75 g of oral glucose. Fasting serum albumin concentrations were also determined.

Of the 78 subjects with IGT, 12 (15.4%, median BMI 29.2 kg/m², median age 56.2 years) became diabetic (DM group), 26 (33.3%, median BMI 27.0 kg/m², median age 49.9 years) reverted back to normal glucose tolerance status (NG group). Over the 4-year

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period a reduction in median BMI was observed: –2.4 kg/m² for the DM group, –1.9 kg/m² for the IGT group and –0.6 kg/m² for the NG group. Table I shows median intakes of nutrients in the three groups. Subjects in all three groups showed low median intakes of nutrients (< 67% of the RDAs). Significantly fewer subjects in the NG group showed low intakes of ascorbic acid ($p = 0.05$) than the DM group. Furthermore, significantly fewer subjects in the NG group showed low intakes of phosphorus ($p = 0.05$), folic acid ($p = 0.004$), ascorbic acid ($p = 0.02$) and vitamin D ($p = 0.05$) than the IGT group. The IGT group showed a significantly lower fat percentage ($p = 0.04$) and lean body mass ($p = 0.04$) than the NG group. Serum albumin levels in the three groups did not differ significantly.

Although the presence of IGT and DM might also have caused suboptimal nutritional status, our results showed that more subjects in the NG group were better nourished with regard to certain micronutrients than the IGT and DM groups. We therefore conclude that optimal nutrition may play a role in the prevention of DM. However, our most disturbing finding was the low reported micronutrient intakes of all three groups, although underreporting in overweight and obese subjects might have occurred. The need for better dietary education and improved standards of living in these communities remains a health care priority.

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**Table I. Median nutrient intakes of the three groups**

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>RDA Males</th>
<th>RDA Females</th>
<th>NG group Males</th>
<th>NG group Females</th>
<th>IGT group Males</th>
<th>IGT group Females</th>
<th>DM group Males</th>
<th>DM group Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A (µg RE)</td>
<td>1 000</td>
<td>800</td>
<td>236</td>
<td>235</td>
<td>154</td>
<td>800</td>
<td>800</td>
<td>235</td>
</tr>
<tr>
<td>Vitamin D (µg)</td>
<td>50</td>
<td>50</td>
<td>1.7</td>
<td>0.58</td>
<td>0.97</td>
<td>800</td>
<td>800</td>
<td>235</td>
</tr>
<tr>
<td>Vitamin E (mg α-TE)</td>
<td>10</td>
<td>8</td>
<td>5.5</td>
<td>4.2</td>
<td>4.1</td>
<td>800</td>
<td>800</td>
<td>235</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>60</td>
<td>60</td>
<td>22</td>
<td>8.8</td>
<td>5.12</td>
<td>800</td>
<td>800</td>
<td>235</td>
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<tr>
<td>Thiamin (mg)</td>
<td>1.2</td>
<td>1.1</td>
<td>0.65</td>
<td>0.54</td>
<td>0.6</td>
<td>800</td>
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<tr>
<td>Riboflavin (mg)</td>
<td>1.3</td>
<td>1.1</td>
<td>0.71</td>
<td>0.62</td>
<td>0.56</td>
<td>800</td>
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<tr>
<td>Niacin (mg NE)</td>
<td>16</td>
<td>14</td>
<td>6.53</td>
<td>3.62</td>
<td>5.04</td>
<td>800</td>
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<td>235</td>
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<tr>
<td>Vitamin B₆ (mg)</td>
<td>1.9</td>
<td>1.7</td>
<td>0.6</td>
<td>0.4</td>
<td>0.5</td>
<td>800</td>
<td>800</td>
<td>235</td>
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<tr>
<td>Folate (µg)</td>
<td>400</td>
<td>400</td>
<td>129.1</td>
<td>91.3</td>
<td>128.1</td>
<td>800</td>
<td>800</td>
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<tr>
<td>Vitamin B₁₂ (µg)</td>
<td>2.4</td>
<td>2.4</td>
<td>2.0</td>
<td>1.11</td>
<td>2.15</td>
<td>800</td>
<td>800</td>
<td>235</td>
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<tr>
<td>Calcium (mg)</td>
<td>800</td>
<td>800</td>
<td>323</td>
<td>294</td>
<td>280</td>
<td>800</td>
<td>800</td>
<td>235</td>
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<tr>
<td>Phosphorus (mg)</td>
<td>800</td>
<td>800</td>
<td>718</td>
<td>506</td>
<td>563</td>
<td>800</td>
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<tr>
<td>Magnesium (mg)</td>
<td>350</td>
<td>280</td>
<td>208</td>
<td>155</td>
<td>178</td>
<td>800</td>
<td>800</td>
<td>235</td>
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<tr>
<td>Iron (mg)</td>
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<td>15</td>
<td>5.6</td>
<td>3.7</td>
<td>5.4</td>
<td>800</td>
<td>800</td>
<td>235</td>
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<tr>
<td>Zinc (mg)</td>
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<td>12</td>
<td>6.7</td>
<td>4.0</td>
<td>4.2</td>
<td>800</td>
<td>800</td>
<td>235</td>
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</tbody>
</table>

RE = retinol equivalents; mg α-TE = tocopherol equivalents; NE = niacin equivalents.