EFFECT OF INTRALUMINAL CONCENTRATIONS ON THE IMPAIRMENT OF GLYCINE ABSORPTION BY GLUCOSE IN THE HUMAN JEJUNUM

G. C. COOK

Department of Medicine, The University of Zambia, Lusaka, Zambia

(Received 31 August 1971)

SUMMARY

1. To investigate the effect of different intraluminal concentrations on the mutual inhibitive effect of glycine and glucose on their jejunal absorption rates, eighteen convalescent Zambian African patients who had no clinical evidence of intestinal disease or of malnutrition were given constant intrajejunal infusions with those solutes either together or alone. A double-lumen tube perfusion system was used, and three solutions containing (A) glycine, (B) glycine and glucose, and (C) glucose, all of which were rendered iso-osmotic with sodium chloride, were perfused in random order at 120 ml/min. The concentration of glycine in the perfusing fluid was either 10 or 20 mM, and that of glucose either 100, 200 or 280 mM. By reference to polyethylene glycol 4000, the absorption rates of the solutes and water were calculated for a 30 cm jejunal segment.

2. At a glucose concentration of 200 or 280 mM, but not 100 mM, the mean rate of glycine absorption was decreased by approx. 30%. Glucose absorption rates were not significantly altered by glycine.

3. These observations, taken in conjunction with those from a previous investigation, are consistent with the view that there are two mechanisms for the jejunal absorption of glycine in man, one of which is inhibited by glucose at high intraluminal concentration.

Key words: glycine, glucose, jejunum, absorption.

The influence of glucose on intestinal amino acid transport in animals has been reported as inhibiting, stimulating or ineffective in different experiments (Newey & Smyth, 1964; Dawson, Newey & Smyth, 1965; Saunders & Isselbacher, 1965; Alvarado, 1966; Bingham, Newey & Smyth, 1966; Chez, Schultz & Curran, 1966; Hardcastle, Newey & Smyth, 1968). These conflicting results could be related to solute concentrations.

Correspondence: Professor G. C. Cook, Department of Medicine, The University of Zambia, P.O. Box 2379, Lusaka, Zambia.
A previous investigation in man \textit{in vivo} has shown that if a solution containing glucose and glycine is perfused in the upper jejunum, there is a significant impairment in the rate of absorption of both solutes compared with the corresponding rates when they are given alone (Cook, 1971a). In that study the concentrations of the solutes in the perfusing fluids were 100 \text{mm}-\text{glycine} and 200 \text{mm}-\text{glucose}. The solution containing both glycine and glucose did not contain sodium and it is possible that that is important in the transport of glycine in man.

In the present investigation the effect of glucose on glycine absorption at various concentrations of both solutes, and the importance of sodium in the perfusing fluid, have been studied in further groups of subjects.

\textbf{SUBJECTS AND METHODS}

Table 1 gives the details of the eighteen Zambian African subjects who were studied. All were male in-patients at the University Teaching Hospital, Lusaka, who were convalescing from a variety of diseases. They agreed to take part after an explanation of the methods and purpose of the study had been made. None had clinical evidence of intestinal disease or of malnutrition. They had not previously been investigated.

Table 1 also gives serum protein and blood haemoglobin concentrations, and results of stool microscopy. Serum total protein and electrophoresis determinations were done as described by Cook (1971b). Subjects 17 and 18 had jejunal biopsies and D-xylose absorption tests (Cook, 1971b); jejunal histology and disaccharidase concentrations were normal for Zambian African patients. The 5 h D-xylose excretion after a 25 g oral load was 2.7 and 1.6 g in those two subjects respectively.

The perfusion technique was described by Cook (1971a). A double-lumen tube was swallowed on the evening before the test. Perusions were done 14–18 h later, after radiological localization of the tube. The perfusing solution was pumped into the upper jejunum at 12.0 \text{ml/min} through the proximal opening and siphonage of the intestinal content was made from the distal opening; the distance from proximal to distal openings was 30 cm. The position of the proximal opening in relation to the ligament of Treitz is shown in Table 1. Table 2 shows the composition of the perfusion solutions. Each subject had perfusions with solutions A, B and C. The solutions contained polyethylene glycol (PEG) 4000 at a concentration of 0.5 g/100 ml and were made iso-osmotic with NaCl where appropriate. Each perfusion was made over a 65 min period; three 10 min collections of siphonage were made during the last 30 min.

The sequence of the perfusions was varied in the different individuals (Table 1) and they all followed directly after one another. In the analysis of data, results previously reported in which six other subjects received perfusions of (A) 100 \text{mm}-\text{glycine}, (B) 100 \text{mm}-\text{glycine} and 200 \text{mm}-\text{glucose}, and (C) 200 \text{mm}-\text{glucose} solutions (Cook, 1971a) are included.

The chemical methods used were described by Cook (1971a). Samples of intestinal siphonage from each 10 min collection during the perfusion of solutions B and C were immediately deproteinized for the glucose oxidase determinations. Samples from all collections were immediately frozen for glycine and PEG determinations. In every case specimens of all perfusion solutions were similarly processed and their contents estimated at the same time as the samples. The glucose and PEG determinations were done in duplicate and the glycine determinations in triplicate. As the presence of glucose gave high readings in the glycine determinations, separate standard curves for glycine in the presence of various concentrations
<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Tribe*</th>
<th>Body weight (kg)</th>
<th>Alcohol</th>
<th>Order of perfusions (see text)</th>
<th>Tube position (cm of proximal opening past ligament of Treitz)</th>
<th>Serum protein (g/100 ml)</th>
<th>Total γ-Globulin (g/100 ml)</th>
<th>Albumin</th>
<th>Hæmoglobin (g/100 ml)</th>
<th>Haemoglobin parasites</th>
<th>Clinical diagnosis</th>
<th>Stool parasites</th>
<th>parasites</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>40</td>
<td>Nsenga</td>
<td>54</td>
<td></td>
<td></td>
<td></td>
<td>2.7</td>
<td>5.0</td>
<td>1.6</td>
<td>148</td>
<td>Nil</td>
<td>Tropical polyarthropathy</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>2</td>
<td>45</td>
<td>Nsenga</td>
<td>54</td>
<td></td>
<td></td>
<td></td>
<td>3.9</td>
<td>1.6</td>
<td>2.0</td>
<td>38</td>
<td>Nil</td>
<td>Bronchiectasis; abdominal pain</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>Tonga</td>
<td>57</td>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>1.8</td>
<td>3.0</td>
<td>127</td>
<td>Nil</td>
<td>Lobar pneumonia; splenomegaly</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>4</td>
<td>49</td>
<td>Tumbuka</td>
<td>48</td>
<td></td>
<td></td>
<td></td>
<td>2.2</td>
<td>4.5</td>
<td>2.5</td>
<td>148</td>
<td>Nil</td>
<td>Undiagnosed</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>5</td>
<td>34</td>
<td>Chikuta</td>
<td>52</td>
<td></td>
<td></td>
<td></td>
<td>1.8</td>
<td>4.6</td>
<td>2.0</td>
<td>148</td>
<td>Nil</td>
<td>Lobar pneumonia; recovered</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>6</td>
<td>71</td>
<td>Seba</td>
<td>56</td>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>3.8</td>
<td>3.0</td>
<td>126</td>
<td>Nil</td>
<td>Mild gastroenteritis (recovered)</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>7</td>
<td>49</td>
<td>Nsenga</td>
<td>52</td>
<td></td>
<td></td>
<td></td>
<td>3.0</td>
<td>2.2</td>
<td>2.0</td>
<td>145</td>
<td>Nil</td>
<td>Lobar pneumonia; recovered</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>8</td>
<td>50</td>
<td>Nsenga</td>
<td>62</td>
<td></td>
<td></td>
<td></td>
<td>1.9</td>
<td>1.0</td>
<td>3.0</td>
<td>145</td>
<td>6</td>
<td>Acute orchitis</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>9</td>
<td>33</td>
<td>Chewa</td>
<td>48</td>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>4.3</td>
<td>2.0</td>
<td>147</td>
<td>1</td>
<td>Lobar pneumonia; recovered</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>10</td>
<td>43</td>
<td>Katonde</td>
<td>79</td>
<td></td>
<td></td>
<td></td>
<td>2.2</td>
<td>4.3</td>
<td>2.2</td>
<td>147</td>
<td>1</td>
<td>Severe abdominal pain</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>11</td>
<td>23</td>
<td>Kalonde</td>
<td>64</td>
<td></td>
<td></td>
<td></td>
<td>1.7</td>
<td>3.8</td>
<td>2.0</td>
<td>147</td>
<td>1</td>
<td>Lobar pneumonia; recovered</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>12</td>
<td>24</td>
<td>Benba</td>
<td>57</td>
<td></td>
<td></td>
<td></td>
<td>1.7</td>
<td>3.5</td>
<td>1.7</td>
<td>122</td>
<td>1</td>
<td>Lobar pneumonia; recovered</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>13</td>
<td>40</td>
<td>Benba</td>
<td>48</td>
<td></td>
<td></td>
<td></td>
<td>1.9</td>
<td>1.9</td>
<td>1.9</td>
<td>151</td>
<td>1</td>
<td>Haematuria due to Schistosoma haematobium infection</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>14</td>
<td>29</td>
<td>Tonga</td>
<td>68</td>
<td></td>
<td></td>
<td></td>
<td>3.3</td>
<td>4.2</td>
<td>3.3</td>
<td>4</td>
<td>Nil</td>
<td>Haematuria due to Schistosoma haematobium infection</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>15</td>
<td>45</td>
<td>Benba</td>
<td>49</td>
<td></td>
<td></td>
<td></td>
<td>2.5</td>
<td>3.0</td>
<td>2.5</td>
<td>137</td>
<td>1</td>
<td>Mild gastritis</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>16</td>
<td>54</td>
<td>Leye</td>
<td>54</td>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>4.3</td>
<td>2.0</td>
<td>8</td>
<td>4</td>
<td>Pulmonary tuberculosis; Urinary tract infection</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>17</td>
<td>51</td>
<td>Mwenyi</td>
<td>51</td>
<td></td>
<td></td>
<td></td>
<td>2.5</td>
<td>4.3</td>
<td>2.5</td>
<td>14</td>
<td>4</td>
<td>Lobar pneumonia; recovered</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>18</td>
<td>46</td>
<td>Angola</td>
<td>60</td>
<td></td>
<td></td>
<td></td>
<td>2.0</td>
<td>4.3</td>
<td>2.0</td>
<td>12</td>
<td>4</td>
<td>Lobar pneumonia; recovered</td>
<td>Nil</td>
<td>Nil</td>
</tr>
</tbody>
</table>

* Confirmation by radiology.

of glucose were constructed. The rates of absorption of glycine, glucose and water were calculated from standard formulae as described by Cook (1971a). In the statistical analysis of results $t$-tests were used.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Solute in perfusion solution</th>
<th>Solute concentration (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Solution A</td>
</tr>
<tr>
<td>1–6</td>
<td>Glycine</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Glucose</td>
<td>—</td>
</tr>
<tr>
<td>7–12</td>
<td>Glycine</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>Glucose</td>
<td>—</td>
</tr>
<tr>
<td>13–18</td>
<td>Glycine</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>Glucose</td>
<td>—</td>
</tr>
</tbody>
</table>

### RESULTS

Table 3 summarizes the reproducibility of the glycine, glucose and water absorption rates during the three 10 min collections of siphonage during each perfusion. The mean and standard deviation during all perfusions for the solutes and water in each subject are given. For the solutes the coefficient of variation is also shown; for water, where the mean is so close to zero, this index is not given.

Effect of glucose on glycine absorption

Fig. 1 summarizes the results. Glucose at a concentration of 100 mM had no significant effect on the rate of absorption of glycine presented to the jejunum as a 10 mM solution (paired $t$-test, 5 degrees of freedom $t = 0.96; P<0.40$). Glucose (200 and 280 mM) however, decreased the rate of absorption of 20 mM-glycine solutions by 25% ($t = 6.46; P<0.01$) and by 33% ($t = 5.72; P<0.01$) respectively. Glucose (200 mM) also decreased the rate of absorption of 100 mM-glycine presented to the jejunum by 33% ($t = 9.92; P<0.001$) (Cook, 1971a).

Effect of glycine on glucose absorption

Fig. 2 summarizes the results. Glycine (10 mM) had no significant effect on the rate of absorption of 100 mM-glucose presented to the jejunum (paired $t$-test, 5 degrees of freedom $t = 2.39; P<0.10$). Glycine (20 mM) had no significant effect on the rate of absorption of glucose at 200 mM ($t = 1.81; P<0.20$) and 280 mM ($t = 0.96; P<0.40$). Glycine (100 mM), however, decreased the rate of absorption of 200 mM-glucose presented to the jejunum ($t = 3.51; P<0.02$) (Cook, 1971a). Subject 18 had a lower glucose absorption rate than the other five subjects in that group (0.13 g min$^{-1}$ 30 cm of jejunum$^{-1}$); his glycine absorption rate was, however, similar to that in the other subjects (0.013 g min$^{-1}$ 30 cm of jejunum$^{-1}$).

Water absorption

Fig. 3 summarizes the results for net water absorption. The highest mean absorption rates were from 100 mM solutions. During the glycine perfusions (A) the rate was significantly higher from the 100 mM than the 10 mM solutions (unpaired $t$-test, 10 degrees of freedom $t =$
TABLE 3. Reproducibility of the results for glycine, glucose, and net water absorption rates during each perfusion in the three groups of subjects investigated

<table>
<thead>
<tr>
<th>Subjects (for composition of the perfusing fluids in these subjects, see Table 2)</th>
<th>Absorption rate of glycine (mg min⁻¹ 30 cm of jejunum⁻¹)</th>
<th>Absorption rate of glucose (g min⁻¹ 30 cm of jejunum⁻¹)</th>
<th>Absorption rate of water (ml min⁻¹ 30 cm of jejunum⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD*</td>
<td>Coefficient of variation (%)</td>
</tr>
<tr>
<td>1-6</td>
<td>6.35</td>
<td>0.52</td>
<td>8.2</td>
</tr>
<tr>
<td>7-12</td>
<td>11.31</td>
<td>0.89</td>
<td>7.9</td>
</tr>
<tr>
<td>13-18</td>
<td>10.74</td>
<td>0.75</td>
<td>7.0</td>
</tr>
</tbody>
</table>

* The SD values are calculated from the formula:

\[ \sigma = \sqrt{\frac{1}{2n} \left( \Sigma x_{it}^2 - \frac{\Sigma x_t^2}{3} \right) } \]

where \( x_{it} \) is the \( i \)th reading for the \( t \)th perfusion, \( x_t \) is the total of the three readings for the \( t \)th perfusion and \( n \) is the number of perfusions.
Fig. 1. Glycine absorption (mg min^{-1} 30 cm of jejunum^{-1}) from the glycine, and glycine and glucose solutions in the subjects studied. The solute concentrations in the perfusing fluids in each group of studies are shown below the horizontal axis. ○, Glycine absorption rate from glycine solution (A); ●, glycine absorption rate from glycine and glucose solution (B). The mean ± 1 SDM are shown for each group of studies.

Fig. 2. Glucose absorption (g min^{-1} 30 cm of jejunum^{-1}) from the glucose, and glucose and glycine solutions in the subjects studied. The solute concentrations in the perfusing fluids in each group of studies are shown below the horizontal axis. ○, Glucose absorption rate from glucose solution (C); ●, glucose absorption rate from glucose and glycine solution (B). The mean ± 1 SDM are shown for each group of studies.
Glycine and glucose absorption in man

5.67; \( P < 0.001 \)). During the glucose perfusions (C) the rate was significantly higher from the 100 mM than the 280 mM solutions (\( t = 3.62; \ P < 0.01 \)). The lowest mean absorption rates were either from solutions with a low (10 or 20 mM-glycine) or high (280 mM-glucose; 280 mM-glucose + 20 mM-glycine; 200 mM-glucose + 100 mM-glycine) solute concentration.

![Graph showing net water absorption (ml min⁻¹ 30 cm of jejunum⁻¹) from glycine (A), glycine and glucose (B), and glucose (C) solutions. The solute concentrations in the perfusing fluids in each group of studies are shown beside the vertical axis. The mean ± 1 SDM are shown for each group of studies.](image)

DISCUSSION

The subjects studied were convalescing from a variety of illnesses. All were relatively well and had no clinical evidence of malnutrition or of intestinal disease at the time of study. Groups of six subjects were investigated in each part of the study and there is no evidence that any of the diseases significantly influenced the conclusions in any of the groups. Many subjects had low serum albumin concentrations. Although the causes of this were not investigated, hypercatabolism of albumin resulting from a recent infection and subclinical malnutrition were probably largely responsible. Previous studies (Cook, 1971a; G. C. Cook, unpublished observation) have indicated that absorption rates of glycine and glucose measured by the same technique as that used in the present investigation are not influenced by serum albumin concentrations.

The present study extends results obtained from an investigation that showed an impairment
of glycine absorption by the monosaccharides glucose and galactose in man in vivo (Cook, 1971a). It is now clear that for a significant impairment of glycine absorption, a concentration of glucose in the perfusing fluid of somewhere between 100 and 200 mM is necessary. The present study also indicates that the inhibition in the rate of glycine absorption reported by Cook (1971a) is not due to a lack of sodium in the perfusing fluid of solution B. With the 20 mM-glycine and 200 mM-glucose (B) solution used in the present study (in which 80 mM-NaCl was also present), the percentage inhibition of glycine absorption by glucose was similar to that observed (Cook, 1971a) when solution B contained 100 mM-glycine and 200 mM-glucose and also in the present study when solution B contained 20 mM-glycine and 280 mM-glucose.

The percentage inhibition of glycine absorption was remarkably constant with 20 mM- and 100 mM-glycine and with 200 mM- and 280 mM-glucose. This cannot be accounted for by a direct overall competitive inhibition between glycine and glucose and must involve a more complex interrelation between the two solutes during absorption from the jejunum. In the rat, experiments suggest that there are two mechanisms for glycine absorption; one is similar to that for other monoamino-monocarboxylic amino acids and another is specific for glycine (Christensen, 1960; Newey & Smyth, 1963). The results of the present study are consistent with the presence of two absorptive processes for glycine in man, only one (accounting for approx. 30% of glycine absorption) of which is inhibited by monosaccharides. The results from subject 18 in whom the glucose absorption rate was low although glycine was rapidly absorbed, suggest that the major part of these two solutes do not share the same transport mechanism. That patient had recently recovered from lobar pneumonia and also had an impairment of D-xylose absorption; impaired glucose absorption rate and a decrease in D-xylose absorption have been demonstrated in the presence of acute and chronic bacterial infections (Cook, 1971b).

In the experimental animal, although an inhibition of amino acid uptake by glucose has been shown in some experiments in vitro, others have shown either no effect, or evidence of stimulation (Newey & Smyth, 1964; Dawson et al., 1965; Saunders & Isselbacher, 1965; Alvarado, 1966; Bingham et al., 1966; Chez et al., 1966; Hardcastle et al., 1968). The reason for these conflicting results is not clear; the present study does not solve the problem but confirms that inhibition is the usual effect in man and at no concentration in the present study was there significant stimulation.

It seems probable that some of the subjects in the present study were marginally malnourished even though there was no clinical evidence of malnutrition. There are only a few studies on the effect of starvation or malnutrition on the absorption of amino acids in man. Adibi & Allen (1970) used a jejunal perfusion technique to show that there is a significant decrease in the absorption rate of several essential amino acids in man after a 2 week fast, and Steiner, Farrish & Gray (1969) used a technique in vitro to demonstrate a decrease in L-valine uptake after similar treatment. Morphological changes in the human jejunum in chronic malnutrition (Stanfield, Hutt & Tunnicliffe, 1965) appear slowly and are not apparent after a 4 week fast (Knudsen, Bradley, Lecocq, Bellamy & Welsh, 1968). The results in the present study suggest that in man marginal malnutrition has very little effect on the rate of glycine absorption since results from different individuals, some of whom were clearly very well nourished, were grouped closely together. In the experimental animal, although an inhibition of amino acid uptake during dietary restriction has been shown in some experiments in vitro (Levin, Newey & Smyth, 1965), others have shown either no effect (Kirsch, Saunders & Brock, 1968; Neale, 1971) or evidence of a stimulation (Kershaw, Neame & Wiseman, 1960; Hindmarsh, Kilby,
Glycine and glucose absorption in man

Ross & Wiseman, 1967; Steiner & Gray, 1969). The rate of glucose absorption has been shown to be decreased by malnutrition in man in vivo, probably in the absence of severe mucosal damage (James, 1968). A confused situation applies to the influence of dietary restriction on glucose absorption in animal experiments. Inhibition in the starved rat (Newey, Sanford & Smyth, 1970), stimulation in the semi-starved rat (Kershaw et al., 1960), and no effect in the hamster (Hindmarsh et al., 1967) have variously been reported.

The present study does not add to a previous suggestion that the inhibition of amino acid uptake by monosaccharides might have a practical importance in man (Cook, 1971a). The greater part of amino acid absorption occurs in the upper jejunum. L-Methionine has been shown to be transported three times more rapidly in the proximal than distal small intestine of man (Schedl, Pierce, Rider & Clifton, 1968) and in a study in vivo in the Zambian African there is evidence that a considerable proportion of L-methionine infused into the upper jejunum reaches the large intestine (Cook, 1972). The intraluminal concentration of glucose required to inhibit glycine absorption as shown in the present study is probably greater than that usually present physiologically. Although Borgström, Dahlqvist, Lundh & Sjövall (1957) demonstrated that after a high glucose meal, glucose concentrations of 28–500 (mean 200) mM are present in the human intestine at the ligament of Treitz, other results suggest that concentrations of 20–40 mM are more likely (Olsen & Ingelfinger, 1968). In the Zambian African subject and in most Africans the diet consists very largely of carbohydrate, in the form of starch and polysaccharide, with marginally adequate concentrations of amino acids (Richards, 1961). The resultant concentration of monosaccharide which comes into contact with the mucosal cell from such a diet is unknown. Whether there is a mutual impairment during absorption between monoamino-monocarboxylic amino acids (other than glycine) and essential amino acids and glucose, is unknown. The present study suggests that marginal malnutrition does not influence the rate of amino acid absorption to any great extent. If, however, overt malnutrition can be shown to impair the absorption rate of amino acids it could enhance the practical importance of the present observation. The relative proportions of dietary protein that are absorbed as amino acid and peptide are not yet known but it seems likely that peptide absorption is of considerable practical importance. A significant impairment of glycylglycine absorption by glucose has recently been demonstrated in man (G. C. Cook, unpublished observation).

In the three groups of subjects the coefficient of variation for the solutes for the three 10 min collections of siphonage during each perfusion was usually less than 10%. The results for net water absorption indicate a much greater variation that is especially marked during the perfusion of solutions with low solute concentrations. The corresponding standard deviations in the subjects who received solutions containing 100 mM-glycine and 200 mM-glucose have been reported (Cook, 1971a) and the means and coefficients of variation can be derived from the values in that paper.

ACKNOWLEDGMENTS

I thank Mr James Tembo for much help with the patients. I also thank Dr N. O. Berg and Dr Arne Dahlqvist for the jejunal histology and disaccharidase concentrations in patients 17 and 18. I am grateful to the Editor of the Journal of Physiology for permission to publish results from my paper in that journal. Financial support for the investigation was provided by The University of Zambia Research Fund.
REFERENCES


