Pancreatic A cell function was studied in a patient with primary adreno-
cortical insufficiency. Plasma glucagon levels in the fasted patient
were found to be 65 and 78 pg/ml, close to the upper limit of our normal
range and did not change after the daily administration of 10 mg of hy-
drocortisone for 4 weeks. The glucagon response to insulin-induced
hypoglycemia was normal. However, the glucagon response to arginine in-
fusion was about one half that observed in normal subjects before treat-
ment. This response became supernormal even if delayed after hydrocor-
tisone treatment.

Relative or absolute glucagon hypersecretion has been reported in every form
of endogenous hyperglycemia so far investigated (6). However, reports on pan-
creatic A cell function in endocrine disorders with a tendency to hypoglycemia
are limited to that of Lawrence (3) who described a case of hypopituitarism with
decreased basal and arginine-stimulated glucagon release.

We measured plasma pancreatic glucagon in a patient with Addison's disease
and found a normal fasting glucagon concentration and a decreased response to
arginine, that became supernormal after treatment with glucocorticoids.

Case Report. T. U., a 47-year -old male, was admitted to Gifu University
Hospital complaining of general fatigue. A diagnosis of Addison's disease was
made and he was treated successfully with hydrocortisone. In January of 1975, at
the age of 52, he came to our clinic for reexamination. His general condition was
good. The skin and buccal mucosa were slightly pigmented. His urine, blood count
and blood chemistry, including serum glucose (98 mg/dl), electrolytes, cholesterol
and urea nitrogen were normal and so was his liver function, except for a serum
γ-globulin of 21%.

Endocrine Function Tests. Endocrine function was studied 4 weeks after
interruption of hydrocortisone therapy. At that time, thyroid function was normal.
A 50 g oral glucose tolerance test gave a relatively flat curve, with a fasting
value of 93 mg/dl, a peak value of 119 mg/dl and a 3 hour value of 83 mg/dl. The serum 11-OHCS concentration was below normal (4.0 μg/dl), showed almost no increase 45 min after the intramuscular injection of 0.25 mg of ACTH, then gradually decreased for the next 75 min. The urinary excretion of 17-OHCS was zero or nearly zero on repeated occasions. As shown in Table 1, before, during and after 3 daily injections of 0.5 mg of long-acting ACTH there was very little increase in urinary 17-OHCS excretion, as compared with that observed in normal subjects or in subjects with secondary adrenal cortical insufficiency.

**TABLE 1**

**URINARY 17-OHCS RESPONSE TO DAILY INJECTIONS OF DEPOT-ACTH FOR 3 DAYS**

<table>
<thead>
<tr>
<th>Day</th>
<th>Patient T. U.</th>
<th>Normal Subjects*</th>
<th>Patients with Secondary Adrenal Cortical Insufficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>control</td>
<td>0.3</td>
<td>3.5 - 6.0</td>
</tr>
<tr>
<td>2</td>
<td>control</td>
<td>0.5</td>
<td>13.0</td>
</tr>
<tr>
<td>3</td>
<td>ACTH</td>
<td>2.0</td>
<td>5.3</td>
</tr>
<tr>
<td>4</td>
<td>ACTH</td>
<td>2.0</td>
<td>21.1</td>
</tr>
<tr>
<td>5</td>
<td>ACTH</td>
<td>2.0</td>
<td>7.4</td>
</tr>
<tr>
<td>6</td>
<td>control</td>
<td>0.5</td>
<td>24.2</td>
</tr>
<tr>
<td>7</td>
<td>control</td>
<td>0.1</td>
<td>8.3</td>
</tr>
</tbody>
</table>

* Values obtained in healthy Japanese subjects.

**Value obtained in a typical case.