Serial Changes in Glucose Utilization and Insulin and Growth Hormone Secretion in Acute Cerebrovascular Disease

BY T. A. HUFF, M.D.,* H. E. LEBOVITZ, M.D., A. HEYMAN, M.D., AND L. DAVIS, B.A.

Abstract:
Serial Changes in Glucose Utilization and Insulin and Growth Hormone Secretion in Acute Cerebrovascular Disease

Serial studies were made of glucose utilization and insulin and growth hormone secretion following intravenous glucose tolerance tests given to 16 patients during recovery from acute cerebrovascular disease. Three groups of patients were identified based on the pattern of change of glucose utilization during the first month of convalescence. A small group showed a persistent defective insulin response to glucose and appeared to have had pre-existing unrecognized adult-onset diabetes mellitus. The two other groups showed either an immediate suppression of glucose utilization or delayed development of glucose intolerance associated with an increase in total insulin secretion. Both of these responses returned to normal by the end of the fourth week. Growth hormone secretion correlated with the severity of the stroke. Alterations in glucose metabolism and insulin secretion seem to occur in most patients following recovery from stroke and undoubtedly reflect transient hormonal or metabolic changes related to either acute stress or tissue injury, depending on the interval after the onset of the vascular episode.

Additional Key Words
hyperglycemia diabetes mellitus cerebral infarction intracranial hemorrhage

Hyperglycemia and glucose intolerance have been recognized as frequent findings in patients with acute cerebral infarction or intracranial hemorrhage.1-5 In some patients this abnormal glucose metabolism is due to diabetes mellitus preceding the stroke and reflects the common association of these two illnesses. In other instances, evidence of a pre-existing or underlying diabetes mellitus cannot be found and the hyperglycemia proves to be only an acute transient disturbance in glucose regulation.

Hyperglycemia and glucose intolerance have been recognized as frequent findings in patients with acute cerebral infarction or intracranial hemorrhage.1-5 In some patients this abnormal glucose metabolism is due to diabetes mellitus preceding the stroke and reflects the common association of these two illnesses. In other instances, evidence of a pre-existing or underlying diabetes mellitus cannot be found and the hyperglycemia proves to be only an acute transient disturbance in glucose regulation.

A number of possible mechanisms for this transient alteration in glucose metabolism can be proposed. (1) It may be caused by the autonomic and hormonal changes which result from the acute stress. (2) It may be due to temporary unmasking of latent diabetes mellitus. (3) It may be the result of metabolic changes secondary to injury of body tissue. (4) It might be due to an irritative effect on glucose-regulating centers in the brain stem or hypothalamus such as has been suggested in a recent study of impaired glucose tolerance in patients with intracranial hemorrhage.6

The present study was designed to determine the frequency, duration, degree and mechanism of the impaired glucose tolerance in patients with acute cerebrovascular disease. It reports the serial changes in glucose utilization and insulin and growth hormone secretion following an intravenous glucose tolerance test given at various times during...
recovery from this illness. Three patterns of altered glucose-insulin dynamics seem to appear during recovery from stroke. One may represent an autonomic or hormonal response to acute stress. The second may be a metabolic response to tissue injury. The third is likely to be the result of previously unrecognized diabetes mellitus.

Case Material and Methods
The case material consisted of 16 patients: 13 had acute cerebral infarction, one had transient cerebral ischemia and two had intracranial hemorrhage. Nine were men and seven were women. Their ages ranged from 36 to 67 years, with an average age of 51 years. All but one of the patients (case 9, with subarachnoid hemorrhage) had sudden onset of monoparesis or hemiparesis associated with other focal neurological deficits. The patients were graded as mild, moderate or severe, depending on the degree of alteration in consciousness following the onset of the stroke. Six patients were considered to have a mild stroke with no alteration in consciousness at any time. One of them had vertebral-basilar insufficiency with recurrent transient cerebral ischemic attacks (case 14). another (case 9, mentioned above) had a relatively benign subarachnoid hemorrhage due to rupture of a congenital berry aneurysm, and the remaining four had cerebral infarction. The moderate group consisted of five patients with cerebral infarction who had clouding of consciousness or confusion for 24 to 48 hours after the onset of the stroke. The remaining five patients (one of whom, case 15, had an intracerebral hemorrhage) were classified as having a severe stroke with considerable alteration in consciousness and required intravenous or nasogastric tube feeding. Only one of the 13 patients with infarction had involvement of the brain stem (case 6). The remaining 12 patients had clinical evidence of unilateral infarction of the cerebral hemispheres with aphasia, visual field defects or tonic conjugate eye deviation to the appropriate side. The presence of infarction of the cerebral cortex in these cases was confirmed by the electroencephalographical, brain scan or arteriographical examinations.

All patients were admitted into the study within five days after the onset of the stroke. Patients were excluded if they were known to have diabetes mellitus, uremia, or recent myocardial infarction, or if they received corticosteroids (for cerebral edema), thiazides, or other drugs known to affect insulin secretion. Four of the patients with severe strokes had received intravenous fluids for two or three days prior to the first measurement of glucose tolerance. One of them continued to have intravenous feedings for an additional two weeks. In these cases, the infusate was changed to normal saline at least four hours before glucose tolerance was measured. In all other patients, oral or tube feedings were withheld overnight prior to testing.

The intravenous glucose tolerance tests (IVGTT) were carried out as previously described. Fasting samples were taken following which 25 gm of glucose was rapidly infused in a two-minute period and samples for glucose, insulin and growth hormone determinations were drawn at five-minute intervals for one hour. A 2.5-minute sample was also taken for insulin.

Blood glucose was measured by a glucose oxidase method. The logarithm of individual glucose values was plotted against time. The slope of the linear segment of the curve was derivable by regression analysis and the regression coefficient (b) was multiplied by a constant (-230.3) so as to express K, the rate of glucose fall in the percent per minute, which represents an estimate of glucose utilization. K values below 1.0 were considered diabetic. The normal K value previously reported from this laboratory is 1.65 ± 0.17 (mean ± SEM).

Plasma immunoreactive insulin (IRI) and growth hormone (IRGH) were measured by the double antibody technique of Morgan and Lazarow, using human insulin and growth hormone standards (obtained from Dr. Mary Root, Eli Lilly Company, and Dr. A. E. Wilhelmi). The area under the insulin response curve was integrated as described previously to allow comparison of total insulin secretion on sequential tests. Growth hormone data were treated the same way. We have previously shown that on repeat studies, control patients show no significant variation in K and growth hormone response, and vary by 6% or less with respect to total insulin secretion.

Each of these 16 patients received three or more intravenous glucose tolerance tests during the 30-day period following their acute cerebrovascular episode. These tests were carried out during three successive stages after the onset of the stroke: Period 1, the first five days after the onset of acute symptoms; Period 2, which extended from the sixth to sixteenth day after the onset of the illness, and Period 3, which extended from the eighteenth to the thirtieth day after the stroke.

Results
The serial changes in glucose disposal constants and insulin and growth hormone secretion following intravenous glucose stimulation for each of the patients in the study are continued...
### TABLE 1
K Values, Insulin Secretion and Growth Hormone Secretion Following 25 Gm Intravenous Glucose During Periods 1, 2, and 3 after Acute Cerebrovascular Disease

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Severity</th>
<th>Period 1</th>
<th>Period 2</th>
<th>Period 3</th>
<th>Total IRH Avg. of all periods</th>
</tr>
</thead>
<tbody>
<tr>
<td>K</td>
<td>Total IRI</td>
<td>IRI Pd 2</td>
<td>K</td>
<td>Total IRI</td>
<td>IRI Pd 3</td>
</tr>
<tr>
<td>Group A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Cl</td>
<td>Moderate</td>
<td>0.48</td>
<td>1699</td>
<td>0.603</td>
</tr>
<tr>
<td>2</td>
<td>Cl</td>
<td>Moderate</td>
<td>0.93</td>
<td>3437</td>
<td>0.696</td>
</tr>
<tr>
<td>3</td>
<td>Cl</td>
<td>Severe</td>
<td>0.64</td>
<td>1362</td>
<td>0.479</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td></td>
<td></td>
<td>0.68</td>
<td>2166</td>
<td>0.593</td>
</tr>
<tr>
<td>Group B</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Cl</td>
<td>Severe</td>
<td>0.71</td>
<td>8595</td>
<td>0.863</td>
</tr>
<tr>
<td>5</td>
<td>Cl</td>
<td>Moderate</td>
<td>0.67</td>
<td>1764</td>
<td>0.834</td>
</tr>
<tr>
<td>6</td>
<td>Cl</td>
<td>Moderate</td>
<td>0.61</td>
<td>3126</td>
<td>1.12</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td></td>
<td></td>
<td>0.66</td>
<td>4495</td>
<td>0.939</td>
</tr>
<tr>
<td>Group C</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Cl</td>
<td>Mild</td>
<td>1.30</td>
<td>2050</td>
<td>1.076</td>
</tr>
<tr>
<td>8</td>
<td>Cl</td>
<td>Mild</td>
<td>1.33</td>
<td>8321</td>
<td>0.718</td>
</tr>
<tr>
<td>9</td>
<td>SH</td>
<td>Mild</td>
<td>1.43</td>
<td>5460</td>
<td>0.981</td>
</tr>
<tr>
<td>10</td>
<td>Cl</td>
<td>Mild</td>
<td>1.46</td>
<td>5536</td>
<td>0.759</td>
</tr>
<tr>
<td>11</td>
<td>Cl</td>
<td>Severe</td>
<td>1.17</td>
<td>7188</td>
<td>0.511</td>
</tr>
<tr>
<td>12</td>
<td>Cl</td>
<td>Severe</td>
<td>1.18</td>
<td>848</td>
<td>0.437</td>
</tr>
<tr>
<td>13</td>
<td>Cl</td>
<td>Mild</td>
<td>1.03</td>
<td>1151</td>
<td>0.379</td>
</tr>
<tr>
<td>14</td>
<td>TIA</td>
<td>Mild</td>
<td>1.01</td>
<td>851</td>
<td>0.454</td>
</tr>
<tr>
<td>15</td>
<td>CH</td>
<td>Severe</td>
<td>1.76</td>
<td>9588</td>
<td>0.872</td>
</tr>
<tr>
<td>16</td>
<td>Cl</td>
<td>Moderate</td>
<td>1.04</td>
<td>4576</td>
<td>0.583</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td></td>
<td></td>
<td>1.27</td>
<td>4557</td>
<td>0.677</td>
</tr>
</tbody>
</table>

Cl — Cerebral infarction, SH — subarachnoid hemorrhage, CH — cerebral hemorrhage, TIA — transient ischemic attacks. Meaning of other abbreviations (K, Total IRI, IRGH, etc.) described in text.
given in table 1. The K value is the glucose disposal constant as defined in the section on Case Material and Methods. Total IRI is the area under the plasma insulin curve for 60 minutes following the glucose stimulation. Previous studies with intravenous glucose tolerance tests in normal patients have shown marked individual variation in both K values and total IRI secretion within the normal population, so that it is not feasible to determine the significance of changes in these parameters by comparing the differences of the mean values where small populations are involved.\textsuperscript{6,8} Since these parameters are relatively constant in the same patient, a more valid analysis may be obtained in a study such as the present one, by using each patient as his own control and analyzing the data by paired differences. Since no data on our patients are available from the pre-stroke period, we have arbitrarily set K values and total IRI in Period 2 as the reference. The total IRI in Period 2 was given an arbitrary value of 1.0 and the total IRI’s in Periods 1 and 3 were compared to it. These data are given in table 1 as:

\[
\begin{align*}
\text{IRI PERIOD 1} & \quad \text{IRI PERIOD 3} \\
\text{IRI PERIOD 2} & \quad \text{IRI PERIOD 2}
\end{align*}
\]

The pattern of insulin secretion in response to glucose was further analyzed by comparing the initial discharge of insulin (that released in the first 20 minutes after glucose administration) to the total insulin secretion (that released during the entire 60 minutes after glucose

\[
\begin{align*}
\text{Table 2} & \\
\begin{array}{cccccc}
\text{Stroke cases} & \text{Period 1} & \text{Period 2} & \text{Period 3} & \text{Period compared} & \text{P values} \\
\hline
\text{Group A} & & & & & \\
1 & .438 & .313 & .578 & 1 versus 2 & \text{NS} \\
2 & .537 & .447 & .794 & 2 versus 3 & \text{NS} \\
3 & .532 & .659 & .621 & 1 versus 3 & <0.05 \\
\text{Mean} & .502 & .473 & .664 & & \\
\pm \text{SEM} & .032 & .100 & .066 & & \\
\text{Group B} & & & & & \\
4 & .359 & .364 & .347 & 1 versus 2 & \text{NS} \\
5 & .304 & .253 & .274 & 2 versus 3 & \text{NS} \\
6 & .380 & .387 & .371 & 1 versus 2 & \text{NS} \\
\text{Mean} & .348 & .335 & .331 & & \\
\pm \text{SEM} & .023 & .041 & .029 & & \\
\text{Group C} & & & & & \\
7 & .427 & .397 & .490 & 1 versus 2 & \text{NS} \\
8 & .552 & .523 & .515 & 2 versus 3 & \text{NS} \\
9 & .293 & .329 & .333 & 1 versus 3 & \text{NS} \\
10 & .518 & .486 & .438 & & \\
11 & .361 & .390 & .307 & & \\
12 & .410 & .405 & .498 & & \\
13 & .377 & .354 & .456 & & \\
14 & .418 & .413 & .445 & & \\
15 & .558 & .497 & .708 & & \\
16 & .654 & .535 & .600 & & \\
\text{Mean} & .457 & .433 & .479 & & \\
\pm \text{SEM} & .035 & .023 & .037 & & \\
\text{Control subjects*} & & & & & \\
1 & .477 & .452 & 1 versus 3 & \text{NS} \\
2 & .583 & .546 & & & \\
3 & .633 & .619 & & & \\
4 & .483 & .524 & & & \\
5 & .547 & .663 & & & \\
\text{Mean} & .554 & .561 & & & \\
\pm \text{SEM} & .030 & .037 & & & \\
\end{array}
\end{align*}
\]

\*Data from reference 6.
SERIAL CHANGES IN GLUCOSE UTILIZATION

administration). These values, designated as

Initial IRI
Total IRI

Total growth hormone secretion for each of the patients was calculated for each of the three studies and then averaged for each patient. These measurements are tabulated as total IRGH and represent an average of all three study periods.

Based on the pattern of change of the K value, three groups of patients were identified. Patients in Groups A and B had abnormally low K values in Period 1. These later became normal in Group A, but remained abnormal in Group B. Group C was characterized by normal K values in Periods 1 and 3, but the K was depressed in Period 2. Figure 1 illustrates these data.

Group A consisted of three patients in whom the K value was initially low (mean 0.68) but improved in Periods 2 and 3. The insulin secretion in response to glucose was maximal in Period 2 and was 59% and 63% of maximum in Periods 1 and 3, respectively. As shown in table 2, the decreased insulin response to glucose in Period 1 was associated with a reduction in the initial discharge of insulin, which accounts for half the total insulin response, instead of two-thirds of the total seen in Period 3 (50.2% versus 66.4%,

p < 0.05). Period 2 would seem to be associated with insulin resistance, since total insulin secretion is maximal, but glucose utilization is still impaired in comparison to Period 3. Figure 2 illustrates the serial changes in glucose utilization, and plasma insulin and growth hormone responses to glucose in a
Group B consisted of three patients in whom the K value remained abnormally low in all three of the study periods. There was no sharp initial discharge of insulin after glucose injection (table 2) and the pattern of insulin secretion during the hour was consistent with that noted in adult-onset diabetic patients. The total insulin secretion during each of the three periods did not differ significantly. The findings in a representative Group B patient are shown in figure 3. These three patients appear to have had pre-existing unrecognized diabetes mellitus, and it is of note that they showed no alteration in glucose utilization or insulin secretion during the evolution of the stroke.

Group C comprised ten patients, the largest number in our study. The initial K values for these patients were all within the normal or borderline range (greater than 1.0) but fell in Period 2, and rose toward their initial value in Period 3. As noted in table 1 and figure 1, insulin secretion was maximal in Period 2, was 68% of maximum in Period 1, and 77.3% in Period 3. Thus Group C patients showed a striking degree of insulin resistance in Period 2 but, unlike the Group A patients, did not show alteration in initial insulin secretion (table 2) in Period 1 or 3 (0.457 versus 0.479, respectively). Figure 4 demonstrates the changes in a representative patient in Group C.

The three groups of stroke patients were separable not only by their K patterns, but also on the basis of significant differences in the degree of acute insulin response to glucose during Period 3. This is shown in table 2 by Initial (0 to 20 minutes) IRI/Total (0 to 60 minutes)/IRI ratios of 0.66, 0.33, and 0.48 for Groups A, B, and C (table 2). In a group of normal subjects, the initial discharge of insulin accounts for over half the total secretory response to rapid intravenous glucose loading as shown by a ratio of initial IRI/Total IRI of 0.545 ± 0.030 with no change in serial studies (table 2). In the Group A patients, this ratio was significantly lower in Period 1 than in Period 3 (0.50 and 0.66, respectively), reflecting impaired initial insulin secretion, and correlated with the abnormal K. Some evidence is available to suggest that glucose utilization (K) is primarily influenced by the initial insulin discharge. In contrast to the Group A patients, no changes in the Initial IRI/Total IRI ratio between Periods 1 and 3 were encountered in Groups B and C. In Group B, the ratio of 0.33 reflects marked impairment of acute insulin release, which has previously been shown to be characteristic of the adult-onset diabetic state.
Serial changes in glucose utilization (K value), plasma insulin and growth hormone responses to intravenous glucose in a representative patient of Group C (Patient No. 16).

No relationship was noted between changes in K value and insulin secretion and the severity of the cerebrovascular episode. Approximately equal numbers of patients with severe and moderate strokes occurred in Groups A, B, and C. However, all patients with mild strokes fell into Group C.

The pattern of growth hormone secretion was quite variable. Resting growth hormone levels were not elevated, but in a sizable number of studies in Period 1 there was a rise in plasma growth hormone following the administration of the glucose. In most patients this rise in growth hormone secretion after glucose tended to decrease after Period 1 but exceptions were frequent, particularly in the moderately and severely affected patients. In four of the cases, total growth hormone secretion exceeded 200 ng-min/ml in Period 3. Groups A, B, and C did not differ significantly with respect to growth hormone secretion (table 1, last column).

However, there was a definite correlation of growth hormone secretion with the degree of severity of the stroke. An average total growth hormone secretion for each patient was obtained by averaging the response in each of the three test periods. Although the changes were modest (a total growth hormone secretion of 200 ng-min/ml represents an average plasma level of 3.7 ng/ml) the mean growth hormone secretion varied directly with the severity of the cerebrovascular episode (fig. 5).

Discussion

Serial intravenous glucose tolerance tests carried out in the first four weeks following acute stroke revealed three patterns of altered glucose-insulin dynamics. Three of the 16 patients (Group B) showed a persistent
defective pancreatic response to glucose characteristic of diabetes mellitus. These three patients probably had previously unrecognized adult-onset diabetes mellitus. In the remaining 13 patients the alterations in glucose-insulin responses were transient and consisted of either an immediate suppression of glucose utilization (Group A—three patients) or delayed development of glucose intolerance associated with an increase in total insulin secretion (Group C—ten patients). Both of these responses returned to normal by the end of the fourth week and undoubtedly reflect transient metabolic and hormonal changes related to the acute cerebrovascular episode. The exact mechanism for these disturbances in glucose regulation is not understood, but several explanations seem possible.

One mechanism for the impairment of glucose utilization which occurred during Period 1 in Group A patients might be the release of catecholamines due to an autonomic response to acute stress. Catecholamines can elevate blood glucose through its effects in increasing hepatic release of glucose and blocking peripheral uptake of glucose. In addition, infusion of pharmacological quantities of epinephrine is known to inhibit insulin release in man. Porte and Bagdade have suggested that there are at least two separate pools of insulin within the pancreatic beta cell: a storage pool which is released immediately upon glucose challenge and a second pool which depends on intact insulin synthetic mechanisms and accounts for continued insulin secretion on exposure to prolonged hyperglycemia. Because the first pool is specifically suppressed by epinephrine, the initial insulin release should be most markedly affected if catecholamines were inhibiting insulin release. In the Group A patients, during the later stages of convalescence (Period 3), the initial insulin release (first pool) accounted for 66% of the total insulin response to glucose. Immediately after the stroke, however, the initial discharge was significantly less, 50%, as one might expect to find if catecholamine secretion were increased at that time. There is some suggestion that increased catecholamine secretion after nonspecific stresses (e.g., surgical operation, severe burns and myocardial infarction with shock) may impair insulin release and result in hyperglycemia. Tomomatsu and his co-workers have reported sudden elevations of catecholamine secretion in patients with intracranial hemorrhage, but only a minimal increase in patients with cerebral infarction. Other workers, however, could demonstrate only occasional acute elevation of urinary catecholamine in patients with intracranial hemorrhage. All of the Group A patients had cerebral infarction and thus are unlikely to have had significant increases in urinary catecholamine secretion. However, we cannot dismiss the possibility that these patients were unusually sensitive to small increases in the circulating catecholamines.

An acute autonomic response to stress could not account for the transient resistance to endogenous insulin observed in Group C patients. These patients developed glucose intolerance two weeks after the onset of stroke, at which time there would be no increase in catecholamine secretion. On the other hand, an alteration in pituitary-adrenal function, such as that observed by Jenkins et al., in patients with subarachnoid hemorrhage might contribute to the insulin resistance in our Group C patients. These workers observed disturbances in the diurnal rhythm of plasma cortisol and abnormal responses to metapyrone and dexamethasone for as long as four weeks after bleeding from an aneurysm of the circle of Willis. No adequate data are available as to the pattern of pituitary-adrenocortical regulation following cerebral thrombosis.

A more likely explanation for our results is the possibility that the insulin resistance and abnormal glucose utilization observed in Groups A and C are related to an hypercatabolic response to injury. In patients, as well as experimental animals with fractures, burns or surgical procedures, marked negative nitrogen balance, mild hyperglycemia and insulin resistance are often found. There are also reports of decreased glucose utilization and increased insulin response to intravenous glucose in the second week after acute myocardial infarction, a pattern similar to that observed in our Group C patients. In patients with severe burns, laboratory evidence of diabetes mellitus and negative nitrogen balance which is reversible only with large doses of insulin have been found to persist for two to three weeks. Although there are no published data on total...
SERIAL CHANGES IN GLUCOSE UTILIZATION

urinary nitrogen excretion after stroke, preliminary data indicate that negative nitrogen balance occurs during convalescence of stroke in the absence of complicating infections. There is also evidence that experimental hemispherectomy in monkeys produces prolonged negative nitrogen balance. The combination of glucose intolerance, insulin resistance and nitrogen loss in patients and animals with brain damage would indicate that acute cerebrovascular disease, such as was seen in our patients, might result in a metabolic response similar to that seen with other tissue injuries.

There have been two recent reports describing impaired glucose tolerance after stroke, but neither of them are entirely comparable to the present study. In the study by Hallpike et al., all of the patients had subarachnoid hemorrhage. The abnormal oral glucose tolerance and mild hyperinsulinemic response observed in his patients during the first week after their hemorrhage usually disappeared when the tests were repeated two to three months later. The patients studied by Kawiak and Stelmasiak showed impaired intravenous glucose tolerance and insulin resistance within three days after acute cerebral hemorrhage or infarction, but the studies were not repeated later in convalescence. It is of interest that neither of the above studies measured glucose responses during the period of convalescence in which we found the most significant changes.

Although a number of animal studies have shown that injury to specific areas of the brain, such as the floor of the fourth ventricle, results in abnormal glucose regulation, there was no evidence in the present study to indicate that disruption of specific brain centers was necessary for alteration in glucose response. The type and location of the brain lesion in our cases varied considerably but seemed to have little effect on the pattern of glucose intolerance. There was likewise no definite relationship between the severity of the stroke as determined by the alteration in consciousness and the changes in glucose metabolism. However, there was a correlation between the alteration in growth hormone secretion and the severity of the stroke. Although there was a small increase in secretion of this hormone observed in our patients with severe stroke, it is unlikely that this was a factor in the development of glucose intolerance and insulin resistance in most of our patients because of the small magnitude of the change in those with mild or moderate strokes.

Acknowledgment

The authors are pleased to acknowledge the expert technical assistance of Miss Sylvia White.

References


551.


17. Huff TA: Unpublished data

Serial Changes in Glucose Utilization and Insulin and Growth Hormone Secretion in Acute Cerebrovascular Disease
T. A. Huff, H. E. Lebovitz, A. HEYMAN and L. Davis

Stroke. 1972;3:543-552
doi: 10.1161/01.STR.3.5.543

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/3/5/543