Glucose metabolism begins or ends with the movement of glucose into and out of cells. Glucose production and utilization involve the movement of substrates through various cycles. The direction and magnitude of this movement are controlled by enzymes whose activity is modulated by acute and chronic regulatory mechanisms. The acute regulation of glucose metabolism occurs through hormone mediated changes in enzymatic activity, principally, the phosphorylation and dephosphorylation of the enzymes. Hormones also exert chronic effects on glucose metabolism by changing the rate of enzyme synthesis. These chronic effects are mediated through alterations in the rate of mRNA synthesis. Under certain metabolic conditions, e.g. fasting or hormonal conditions that favour gluconeogenesis, i.e. low insulin and high glucagon, the low rate of glucose phosphorylation results in intracellular glucose accumulation and glucose is exported by facilitated diffusion into the extracellular fluid (ECF). When hormonal conditions favor glucose utilization, i.e. high insulin and low glucagon, intracellular glucose is rapidly phosphorylated and glucose entry is facilitated along a downhill gradient.

Perturbations of glucose metabolism are characteristic of several responses of pathological conditions. These perturbations include hyperglycemia, as well as modifications in the rates of glucose production and utilization. In general terms, the hyperglycemia must be the result of an increased rate of glucose production relative to changes in glucose utilization. Associated with changes in glucose metabolism are changes in glucoregulatory hormones. The elevation in glucose production is hypothesized due to an increase in
glucagon, along with an increase in epinephrine and cortisol. Shamoon et al. (1981) found a synergistic effect of these three hormones which markedly accelerate gluconeogenesis. Therefore, increased plasma levels of these hormones which counterregulate insulin could contribute significantly to the metabolic disturbances characteristic of the response to the pathology of the autonomic nervous system (ANS).

Historically, the parasympathetic and the sympathoadrenal systems through their neurotransmitters and hormones are thought to work in concert to maintain glucose homeostasis. However, the physiologic interactions of these on protein and amino acid homeostasis have not been closely examined. During euglycemia, insulin is known to decrease plasma amino acid concentration, primarily through its inhibition of protein breakdown and via an increased intracellular amino acid transport (Lundholm et al., 1981; Flakoll et al., 1989). Conversely hyperglycemia has been demonstrated to influence protein metabolism by increasing protein breakdown (Rice et al., 1994). The opposing actions of these dominating factors that regulate protein metabolism would suggest that their concerted action to maintain protein homeostasis is important during dynamic physiologic conditions such as stress and mealtime, as well as during pathological conditions such as diabetes and trauma.

Under ordinary conditions, the most likely role of stimulated protein breakdown is the maintenance of amino acid pools between meals (Scornik, 1984). Under pathological conditions such as uncontrolled diabetes, surgical stress, trauma, burns, sepsis, there is elevated protein catabolism, loss of lean tissue protein stores, stimulated gluconeogenesis, enhanced amino acid oxidation and escalated urea synthesis (Flakoll et al., 1994). Therefore, in the present study protein metabolism was studied in the kidney to observe the effects of autonomic manipulation on the total protein content and also transaminases, the enzymes involved in manufacturing glucose from protein sources.

Evidence has been accumulated to show that the basic underlying mechanism of defective glucose utilization by tissues in diabetes mellitus is the basement membrane alteration associated with this pathological condition (Passmore and Eastwood, 1986). It was therefore felt that defective membrane function can be assessed in terms of membrane bound marker enzymes such as Acid phosphatase, Alkaline phosphatase and Na⁺ K⁺ ATPase, which
are thought to be an essential part of plasma membrane of animal cells and play an important role in active transport of many organic and inorganic ions.

Since the autonomic nervous system is implicated in the regulation of gluconeogenesis via amino acid metabolism, the present investigations were conducted on vagotomized and adrenalectomized rats to scrutinize the total protein content of the renal cells and the activities of the enzymes involved in transamination as well as those concerned with transport and uptake of glucose.

MATERIAL AND METHODS
Male albino rats (*Rattus norvegicus albinus*) of Charles Foster strain weighing around 150-200 g were used for the study. The animals were acclimatized at standard laboratory conditions with food and water *ad libitum*. They were divided into three groups and subjected to different surgical operations.

I  VAGOTOMY (VGX)  
SHAM VAGOTOMY (VGS)

II  ADRENALECTOMY (ADX)  
SHAM ADRENALECTOMY (ADS)

III  VAGOTOMY + ADRENALECTOMY (VGX + ADX)  
SHAM VAGOTOMY + SHAM ADRENALECTOMY (VGS + ADS)

Overnight fasted animals of both experimental and control groups were sacrificed 48 hrs after the operations. The kidney was excised, blotted free of blood and used for preparing various homogenates to carry out the following estimations:

Protein was estimated by the method of Lowry *et al.* (1951) and expressed as mg/100 mg wet tissue. Acid phosphatase and alkaline phosphatase were estimated by the method of Linhardt and Walter (1963), and the activity was expressed as μ moles PNP released/mg protein/ 30 min. Aspartate and alanine transaminase were estimated by the method of Bergmeyer and Bernt (1963) and expressed as Karmen units/mg protein/min. Na⁺-K⁺-ATPase was estimated by the method of Post and Sen (1967) and expressed as μg phosphorous released /mg protein/10 min.
Table 3.1  Protein, transaminases and transport enzymes in rat kidney after vagotomy, adrenalectomy and vagotomy in combination with adrenalectomy.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Protein (mg/100 mg Wet Tissue)</th>
<th>Aspartate Aminotransferase (Karmen units/mg Protein/Min.)</th>
<th>Alanine Aminotransferase (Karmen units/mg Protein/Min.)</th>
<th>Acid Phosphatase (μM PNP released/mg Protein/30 Min.)</th>
<th>Alkaline Phosphatase (μM PNP released/mg Protein/30 Min.)</th>
<th>Na⁺-K⁺-ATPase (μg P released/mg Protein/10 Min.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sham</td>
<td>Experimental</td>
<td>Sham</td>
<td>Experimental</td>
<td>Sham</td>
<td>Experimental</td>
</tr>
<tr>
<td>Vagotomy</td>
<td>24.92±1.56*</td>
<td>19.22±1.01**</td>
<td>22.90±0.97</td>
<td>25.88±1.08**</td>
<td>24.01±0.75</td>
<td>20.35±1.16</td>
</tr>
<tr>
<td>Adrenalectomy</td>
<td>24.01±0.75</td>
<td>22.90±0.97</td>
<td>25.88±1.08</td>
<td></td>
<td>25.60±1.37</td>
<td></td>
</tr>
<tr>
<td>Vagotomy + Adrenalectomy</td>
<td>20.35±1.16*</td>
<td>19.22±1.01**</td>
<td>22.90±0.97</td>
<td>25.88±1.08**</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Values are expressed as mean ± SEM of 6 experiments; * p<0.05; ** p<0.02; *** p<0.01; **** p<0.001.
Table 3.2 Percentage change (compared to controls) in protein, transaminases and transport enzymes in the kidney of rats subjected to vagotomy, adrenalectomy and vagotomy in combination with adrenalectomy.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Vagotomy</th>
<th>Adrenalectomy</th>
<th>Vagotomy + Adrenalectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein (mg/100 mg Wet Tissue)</td>
<td>23**</td>
<td>13**</td>
<td>15*</td>
</tr>
<tr>
<td>Aspartate Aminotransferase (Karmen units/mg Protein/ Min.)</td>
<td>22***</td>
<td>15***</td>
<td>15*</td>
</tr>
<tr>
<td>Alanine Aminotransferase (Karmen units/mg Protein/ Min.)</td>
<td>47***</td>
<td>37***</td>
<td>21**</td>
</tr>
<tr>
<td>Acid Phosphatase (μM PNP released/mg Protein/30 Min.)</td>
<td>16*</td>
<td>16*</td>
<td>15*</td>
</tr>
<tr>
<td>Alkaline Phosphatase (μM PNP released/mg Protein/30 Min.)</td>
<td>20**</td>
<td>41***</td>
<td>14*</td>
</tr>
<tr>
<td>Na⁺-K⁺-ATPase (μg P released/mg Protein/10 Min.)</td>
<td>43***</td>
<td>32***</td>
<td>20**</td>
</tr>
</tbody>
</table>

*Values corrected to nearest whole number; * p< 0.05; ** p< 0.02; *** p< 0.01; **** p< 0.001
Figure 3.1 Level of Protein and activity of Acid Phosphatase in kidney of rats subjected to vagotomy (VGX), adrenalectomy (ADX) and their combination (VGX + ADX)

(A)

(B)

* p < 0.05; ** p < 0.02.
Figure 3.2 Activities of Aspartate and Alanine Amino Transferase in kidney of rats subjected to vagotomy (VGX), adrenalectomy (ADX) and their combination (VGX + ADX)

(A)

* p < 0.05; ** p < 0.02; *** p < 0.01.

(B)
Figure 3.3 Activities of Alkaline Phosphatase and Na^+\textsuperscript{+}-K^+\textsuperscript{+}-ATPase in kidney of rats subjected to vagotomy (VGX), adrenalectomy (ADX) and their combination (VGX + ADX)

\begin{figure}
\centering
\begin{subfigure}{\textwidth}
\centering
\includegraphics[width=\textwidth]{figure3a.png}
\caption{Alkaline Phosphatase}
\end{subfigure}
\begin{subfigure}{\textwidth}
\centering
\includegraphics[width=\textwidth]{figure3b.png}
\caption{Na^+\textsuperscript{+}-K^+\textsuperscript{+}-ATPase}
\end{subfigure}
\end{figure}

* p < 0.05; ** p < 0.02; *** p < 0.01.
Figure 3.4 Percentage change in renal protein content and the activities of transport enzymes and transaminases after vagotomy (A), adrenalectomy (B) and their combination (C).
Statistical analysis:
Statistical analysis was done by employing Student’s t-test to determine the significance of the data. P < 0.05 was considered to be significant.

RESULTS (Table 3.1, 3.2, Figures 3.1 to 3.4)
In the kidney of the vagotomized rats, protein content was reduced considerably (23 %, p < 0.02), whereas the adrenalectomized rats manifested an increase in the protein content (13%). The VGX + ADX rats showed reduced protein content, however, the degree of this decrease (15 %) was not as much as in the VGX rats.

Both aspartate and alanine amino transferases showed enhanced activities after vagotomy, the rise being significant in both the enzymes (22 % and 47 % increase respectively). Adrenalectomized rats showed a significant decline (15 % and 37 % respectively, p < 0.01) in the activities of these enzymes in the kidney. In the VGX + ADX rats however, these enzymes manifested a dissimilar trend, AST increasing by 15 % and ALT decreasing by 21%.

Vagotomized rats showed a slightly increased activity of acid phosphatase in the kidney (16%) contrary to the adrenalectomized and VGX + ADX rats, where a decrease was noticed (16 % and 15 %, p < 0.05).

Alkaline phosphatase activity was seen to be reduced by 20 % in the kidney of rats subjected to vagotomy, unlike in the adrenalectomized rats where a notable rise of 41 % (p < 0.01) was found. In the VGX + ADX rats again a reduced activity was observed (14 %).

Na⁺-K⁺-ATPase activity manifested a rise of 43 % (p < 0.01) in the kidney of vagotomized rats, whereas a reduced activity (32 %) was observed in the adrenalectomized rats. In the VGX + ADX rats, again a small rise of 20 % was seen in the activity of this enzyme.

DISCUSSION
The decrease in the protein content observed in vagotomized rats indicates enhanced protein breakdown caused after cholinergic denervation. In the present experimental group, absence
of vagally mediated enhancement of insulin secretion, following a vagal nerve section would result in a diminished insulin release and therefore an abnormal metabolic response. Vagal input plays a functionally important role in the control of insulin secretion (Campfield and Smith, 1983). In turn, protein degradation is strongly influenced by amino acids, insulin and other regulatory factors. Amino acid and insulin deprivation normally accelerates protein breakdown, whereas excess amino acid and insulin have opposite effects (Mortimore and Schworer, 1980). Increased rates of gluconeogenesis usually occur when there are low circulating levels of insulin (Davis et al., 1995). In underinsulinized diabetic individuals, unrestrained gluconeogenesis contributes significantly to the elevated fasting hyperglycemia (Wahren et al., 1972). Moreover, glucagon stimulates protein breakdown (Mortimore and Ward, 1976). Therefore, the present decline in the kidney protein content could be attributed to the lowered insulin secretion, due to vagal section, as well as due to the possibly elevated sympathetic tone enhancing glucagon release.

Adrenalectomized rats showed an increase in the protein content of the tissue. Glucocorticoids inhibit protein synthesis, and at higher concentration may even stimulate protein degradation (Bolander et al., 1981), the resulting amino acids serving as precursors for glucose (Goldstein et al. 1995). Elimination of glucocorticoids following adrenalectomy would lead to the attenuation of their catabolic effects on protein, and hence this rise in the total protein content of the kidney. Also, norepinephrine enhances gluconeogenesis by stimulating precursor release by its catabolic actions and increasing substrate movement from the periphery (Connolly et al., 1991). Loss of this regulator after ADX would also lead to an increase in the total protein content.

The animals with VGX + ADX showed a diminished renal protein content. Cholinergic activity is impaired as a result of vagotomy. This results in reduced insulin secretion (Yadav, 1997), which in turn could lead to protein breakdown. Performing adrenalectomy in the same animals, however failed to stop the protein catabolism, although ADX would eliminate the adrenal glucocorticoids and catecholamines that are responsible for protein breakdown. Thus, the effect of vagotomy is more prominently manifested in the animals with simultaneous operations for vagotomy and adrenalectomy.
The physiological significance of protein metabolism can be assessed by estimating enzyme activities concerned with these mechanisms. The transaminases are generally known to have a role in amino acid metabolism. They carry out the conversion of amino acids to precursors of glucose such as oxaloacetate and pyruvate.

Aspartate aminotransferase, a ubiquitous pyridoxal phosphate dependent enzyme, plays a major role in amino acid metabolism, and constitutes a link between the urea and the TCA cycles (Cooper and Meister, 1985). In the present study, the VGX animals manifested an increase in the activity of this enzyme in the kidney. Insulin is known to decrease the AST transcription, conversely, diabetes is known to increase its activity in the liver (Pave-Preux et al., 1990). Vagotony would lead to a decrease in insulin secretion (Yadav, 1997). Therefore, the rise in the activity of this enzyme could be because of lack of insulin after cholinergic denervation. This would result in an accelerated gluconeogenic rate.

Adrenalectomized rats showed a decreased activity of this enzyme, indicating no transamination taking place from aspartate in this condition. Glucocorticoids specifically regulate AST activity in rat liver and kidney by inducing AST activity, this effect being potentiated by cAMP and inhibited by insulin (Pave'-Preux et al., 1990). Synthetic glucocorticoid dexamethasone has been shown to increase the AST mRNA (Pave-Preux et al., 1990; Feilleux-Duche et al., 1994). Adrenalectomy would cause a loss of the glucocorticoids (Chapter 6), and hence the decline in the activity of this enzyme.

In VGX + ADX animals, however, the activity of this enzyme again showed a rise. This indicates that adrenalectomy could not override the increased transamination from aspartate produced by vagotomy.

The other transaminase, alanine aminotransferase, facilitates the conversion of alanine to pyruvate, which in turn can be converted into glucose. The increase in the activity of this enzyme after vagotony indicates the activation of the gluconeogenic pathway and confirms the inhibitory effect that insulin has on gluconeogenesis, which is released due to vagal denervation. An elevated sympathetic tone resulting in an increased glucagon secretion after vagotony could also lead to this effect. Boden et al (1977) have explained that glucagon
aids in amino acid disposal by increasing intracellular transport of amino acids including
alanine, as well as their conversion to glucose, probably acting via cAMP.

Adrenalectomy caused a decline in the activity of this enzyme, indicating a low rate
of transamination from alanine to pyruvate. This would also favor hypoglycemia. An acute
rise in plasma cortisol might have detectable effects on carbohydrate metabolism. Shamoon
et al. (1980) and Goldstein et al. (1995) have demonstrated acute hypercortisolemia to
decrease glucose clearance, leading to an increase in plasma glucose level. Glucocorticoid
hormones cause the induction of ALT (Rodwell, 1985). Goldstein et al. (1995) have reported
increased gluconeogenic conversion of alanine into glucose due to hypercortisolemia.
Dexamethasone has been shown to stimulate renal gluconeogenesis (Marija and Andreis,
1994).

In the VGX + ADX rats, again a decrease was observed in the activity of ALT. It
seems that the rise in the activity of this enzyme caused by vagotomy could have been
suppressed by performing adrenalectomy. Stimulation in the activities of AST and ALT
obviously indicates an increased utilization of alanine and aspartate as source of oxidative
energy. An efficient processing of alanine and aspartate produced as a result of protein
degradation is indicated. But in the VGX + ADX rat kidney, there is a decrease in the activity
of ALT and an increase in the activity of AST. This indicates the utilization of aspartate but
not alanine in the gluconeogenic conversion. The proteins thus provide a continuous supply
of amino acids which serve to participate in gluconeogenesis. Vagal impairment may elevate
sympathetic tone which in turn, along with glucagon and adrenal glucocorticoids would
increase gluconeogenesis. Hence cholinergic denervation may lead to relative
hyperglucagonemia and increased availability of glucogenic amino acids. These factors could
combine and as a consequence, could result in suppressing the effects of insulin on glucose
uptake. In the present study however, only a partial effect of this is seen, as only one of the
two transaminases studied is activated.

Transport enzymes such as acid phosphatase, alkaline phosphatase, and Na⁺-K⁺-
ATPase are of major biological importance within the kidney. Acid and alkaline phosphatases
as phosphomonoesterases, subserve a variety of processes requiring the mobilization of
phosphate radicals or entail dephosphorylation as steps in anabolism, catabolism and transport

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aids in amino acid disposal by increasing intracellular transport of amino acids including 
alanine, as well as their conversion to glucose, probably acting via cAMP.
of ions (Moog, 1946). They also aid in the biosynthesis of mono and dinucleotides, metabolism of carbohydrates, proteins and lipids and in growth and differentiation processes (Moog, 1946; Bradfield, 1950). Inactivation of these enzymes would tend to alter both the function and the viability of kidney tubules. Therefore, they can be used as indicators of normal and impaired renal function.

Though the function of acid phosphatase in the kidney is somewhat evasive, the organ is a fairly rich source of this enzyme, being present in glomeruli and throughout the tubule system into the excretory ducts (Moog, 1946). In the present experimental setup, the vagotomized rats showed an increased activity of this enzyme. Acid phosphatase is a part of the lysosomal-vacuolar system, which is generally believed to play a major role in the digestion of intracellular proteins in many mammalian cells (Mortimore and Poso, 1984). Thus, a reduction in the activity would indicate an accelerated protein degradation. This degradative state is amenable to the lowered protein content of kidney after vagotomy. This protein catabolism could provide the renal cells with needed amino acids. In several tissues, insulin inhibits protein degradation by both inhibiting autophagic lysosomal vacuole formation and the inactivation of acid phosphatase (Jefferson et al., 1974). Therefore vagotomy would lead to an increase in the activity of this enzyme as a consequence of withdrawal of insulin after parasympathetic denervation. Sneer et al. (1970) have shown a similar rise in the activity of acid phosphatase in alloxan diabetic rats.

In both ADX and VGX + ADX, a decrease was seen in the activity of acid phosphatase. Adrenal steroids and catecholamines have a protein catabolic action (Connolly et al., 1991), while adrenal corticosteroids induce gluconeogenesis (Munck et al., 1984). Therefore elimination of these groups of hormones would have a negative effect on the enzymes involved in protein degradation such as the acid phosphatase, which is reflected in the present study. The effect of insulin withdrawal seems to have been surmounted by ADX, as can be observed from the reduced activities of this enzyme in both, ADX singly and in combination with VGX.

After vagotomy, activity of alkaline phosphatase decreased in the renal tissue, indicating a reduced uptake activity. Alkaline phosphatase is present in a high concentration in the kidney cortex, and is concerned with reabsorption of sugar (Moog, 1946). It has been
found most highly concentrated in the proximal convoluted tubules (PCT) and especially in
the brush borders (Moog, 1946). Thus, tubular dysfunction could be predicted from the loss
of the activity of this enzyme because it is integrally concerned in renal function. In the brush
border segments of proximal tubules of renal cortex, alkaline phosphatase plays an integral
role in kidney function through the reabsorption of glucose molecules from the tubules by
phosphorylated mechanisms (Moog, 1946; Bradfield, 1950). Capaldo et al. (1992) have
shown that epinephrine antagonizes insulin induced stimulation of glucose uptake. Vagotomy
led to a decreased activity of alkaline phosphatase. Removal of parasympathetic tone could
antagonistically influence the sympathetic tone by enhancing it. There would thus be a
decreased uptake of glucose in the renal tubules.

Adrenalectomy resulted in an increase in the activity of alkaline phosphatase in the
kidney. Glucocorticoids impair whole body glucose uptake and oxidative and non oxidative
glucose disposal in vivo (Nosadini et al., 1983; Baron et al., 1987; McMahon et al., 1988).
Adrenalectomy removes the glucocorticoid hormones and in turn their inhibitory action on
blood uptake. Moreover, adrenalectomy would also reduce epinephrine and thus release its
inhibitory effect on insulin induced glucose uptake. Therefore, increased activity of alkaline
phosphatase could facilitate increased glucose uptake by brush border of PCT.

Rats with VGX+ ADX again showed a decrease in the activity of this enzyme, which
indicates a decreased uptake of glucose. This effect is like vagotomy, indicating that the
parasympathetic system has a major control over this enzyme and in turn, over glucose
uptake.

Another important phosphatase involved in transport and uptake is the Na⁺-K⁺-
ATPase. It is a ubiquitous enzyme responsible for regulating cell ionic composition and
volume in many tissues (Ilio and Hess, 1992), being involved in cotransport of glucose and
amino acids along with Na ions across plasma membrane (Krishnakumari and Govindarajulu,
1991). It is present in high concentration in the kidney where the pump is believed to be
responsible for reabsorption glucose, amino acids and cations like Na⁺, Ca²⁺ and Mg²⁺
(Kumthekar and Katyare, 1992)
In the vagotomized animals, Na⁺-K⁺-ATPase enzyme showed a notably increased activity. This seems to depart from the reports showing Na⁺-K⁺-ATPase and sodium pump activities to be stimulated by insulin in a variety of tissues including the kidney (DeFronzo, 1981; Fidelman et al., 1982). Paradoxically, however, in the present study of the VGX animals, where insulin insufficiency is created by cholinergic denervation, the activity of this enzyme in the kidney increased. Similar elevation in the activity has also been demonstrated in the cortical or outer medullary region of the kidney in streptozotocin (STZ) diabetic rats (Ku et al., 1986). In the diabetic state, the kidneys are constantly exposed to increasing levels of circulating glucose (Maker et al., 1975). Among the renal physiological consequences of diabetes are increased glomerular filtration rate (GFR), osmotic diuresis and increased Na⁺-linked glucose delivery to the renal tubules. It has been proposed that increased activity of Na⁺-K⁺-ATPase resulting from STZ diabetes would be a direct influence of the increased GFR in this state, which would result in increased delivery of Na⁺ to renal tubules, thus stimulating Na⁺ resorptive process (Fine and Bradley, 1985; Nord et al., 1986). The immediate consequence of increased Na⁺ uptake at the luminal border of the tubules is an elevation of intracellular Na⁺ which stimulates the Na⁺ pump at the basolateral surface of the tubule. In diabetes, increased delivery of glucose to the tubules is expected to promote increased Na⁺ glucose cotransport. The sequence of physiological events may contribute to acute and chronic elevation of renal Na⁺-K⁺-ATPase activity after the induction STZ diabetes. Increased glucose delivery to the tubules is a consequence of the elevated plasma glucose concentration in an insulin deficient state, which follows vagotomy in the present study, and is also analogous to diabetic condition.

Adrenalectomized rats showed a decreased activity of Na⁺-K⁺-ATPase in the kidney. It has been found that glucocorticoids can augment Na⁺-K⁺-ATPase activity in the kidney (Rayson and Lowther, 1984). Therefore, adrenalectomy could be accountable for the decline in the activity of this enzyme. Na⁺-K⁺-ATPase activity has been shown to be decreased after adrenalectomy in medullary collecting tubules (MCT) and cortical collecting tubules (CCT) (El Mernissi and Doucet, 1983), which can be restored by glucocorticoid or aldosterone administration (Klein and Lo, 1992).
In animals with VGX + ADX, the activity of this enzyme increased, indicating an effect parallel with vagotomy. Thus it can be interpreted that the parasympathetic system has a principal role in the control of this enzyme.

To sum up the protein metabolic picture that the kidney presents after vagotomy, it can be stated that an enhanced proteolysis and in turn increased transamination add up to the hyperglycemia (Chapter 2) by facilitating the conversion to glucose. Also a reduced uptake can be inferred. In the adrenalectomy, a reversal of the profile is observed, with the proteolysis as well as transamination being reduced. The uptake mechanism however increased. This is amenable with the hypoglycemia of this condition as discussed earlier (Chapter 2). In the VGX + ADX rats the proteolysis and the transamination produced by vagotomy could be brought under check, though not having favorable effect on the uptake mechanism. Thus it can be concluded that adrenalectomy is able to counter the accelerated protein metabolism encountered in vagotomy by controlling the proteolytic and transamination pathways.