"Not everything that counts can be counted and not everything that can be counted, counts"

- Albert Einstein

Conclusions
The following conclusions can be drawn from the results obtained:

I. Relationship between blood glucose level and small intestinal transit

1. Physiologically-induced hypoglycemia by short and long periods of food deprivation have not altered the SIT. However, the drug-induced fall in blood glucose was associated with acceleration of SIT.

2. Physiological glycemic agent (dextrose) could attenuate the SIT only at very high dose.

3. Maintenance of glycemic state by intermittent dextrose administration has not altered the SIT.

4. Clonidine-induced elevation of blood glucose is associated with attenuation of SIT.

5. Metoclopramide-induced acceleration of SIT is associated with elevation of blood glucose level.

6. Atropine-induced attenuation of SIT is not associated with any alteration in glycemic state.

These findings indicate that altered glycemic states do not influence the changes in SIT, except severe hyperglycemic state or drug-induced effects.

II. Relationship between insulin level and SIT

1. Short or long periods of fasting-induced insulin levels have no effect on SIT. Normal insulinemic state might be involved in maintaining the physiological intestinal transit.

2. Oral hypoglycemic drug glibenclamide produced significant acceleration of SIT associated with elevation of serum insulin level. A sudden fall in BG levels or elevation of serum insulin level may also be involved in acceleration of SIT.
3. Mild to moderate hyperglycemic condition has no effect on motility of small intestine. The reason may be that these states produced attenuation of GI motility, might be counteracted by simultaneously elevated serum insulin level, which lead to normal SIT. But, in severe hyperglycemic condition, direct hyperglycemic effect on SIT dominates over insulin effect and attenuation of SIT becomes evident.

4. Clonidine per se attenuated the SIT. It was also associated with reduced level of insulin. We postulate that attenuation of GI motility is at least in part due to reduction in serum insulin.

5. Under the influence of prokinetic drug (metoclopramide) a moderate elevation of insulin level may play favourable role in acceleration of SIT.

6. However, under the influence of strong depressant on GI motility (atropine), a slight elevation of insulin level fail to normalize the SIT.

These findings indicate insulinenic state does not possess stronger effect on SIT, and is liable to be affected by stronger physiological or pharmacological agents.

III. Effect exogenous insulin on SIT

1. The significant finding of this study is, insulin administration at lowest dose accelerated SIT without affecting blood glucose. Insulin, like antinociception, exhibit inherent acceleratory effect on SIT.

2. In normal animals insulin administration elevated serum insulin level which might be involved in potentiating acceleration of SIT which is further supported by elevated C-peptide levels.

IV. Stimulatory effect on GI motility by insulin is free from development of tolerance.
V. The following mechanisms can be deduced in accelerating effect of insulin on SIT- α-adrenergic pathways > Cholinergic pathways > Calcium channels.

VI. Centrally administered insulin has accelerated SIT only at higher doses. This indicates acceleration of SIT is the major responsibility of peripheral insulin and minor to central pathways.

VII. Insulin in vitro produced tonus of the longitudinal muscle of the ileum which gives direct evidence for myogenic activity of insulin.

All these observations in this study collectively favours to conclude that peripheral insulin from exogenous source accelerates the motility of small intestine.

The findings of this experimental study can be further validated in humans by studying the gastrointestinal complications associated with untreated diabetes and any such complications associated with diabetic patients when treated regularly with insulin can give the insight whether the therapy worsens the diarrhoea or corrects the previously associated constipation.