SUMMARY AND CONCLUSION
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Experimental lesions of the gastric and duodenal mucosa have been employed to study the anti-ulcer activity of calcium channel blockers. These studies were undertaken to provide some rationale for their reported anti-ulcer activity in animals and human beings. All the three calcium channel blockers (verapamil, nifedipine and diltiazem) have been found to be effective in different models of experimentally induced gastric ulcers.

Verapamil has been found to be effective in reducing gastric ulceration at all the doses. However, higher dose range was required for the protective activity against cysteamine-induced duodenal ulcers. Nifedipine was effective in both gastric and duodenal ulcer models at higher doses. Diltiazem was effective only in gastric ulcer models at all the dose level studied.

Following are the findings of the present study:

(a) Verapamil and diltiazem significantly reduce volume of gastric content in aspirin treated pylorus ligated rats.
(b) Verapamil and diltiazem significantly reduce total acidity in aspirin treated pylorus ligated rats.
(c) Verapamil and nifedipine significantly reduce score for intensity in cysteamine induced duodenal ulcer model at higher dose level i.e. 32 mg/kg.
(d) Verapamil, diltiazem and nifedipine significantly reduce ulcer index in all the three models of gastric ulcers viz. aspirin induced gastric lesion model, pylorus ligation model and aspirin treated pylorus ligated ulcers in rats.
(e) The calcium channel blockers under study prevent mucus loss as evident from the rise observed in stomach wall mucus content against aspirin treated pylorus ligated rats.

(f) The calcium channel blockers significantly increase total carbohydrate content of gastric juice in aspirin treated pylorus ligation model.

(g) The calcium channel blockers significantly reduce protein content of gastric juice against aspirin treated pylorus ligated rats.

(h) The calcium channel blockers significantly increase TC/PR ratio leading to enhancement of mucin activity.

In conclusion, verapamil and nifedipine produce significant anti-ulcer activity in gastric and duodenal ulcer models. Diltiazem showed significant gastroprotective effect against only gastric ulcer models. The action of these drugs cannot be attributed to the modification of acid pepsin activity. On the basis of these observations, it is suggested that these drugs increase TC/PR ratio and gastric wall mucus content which may be responsible for strengthening the mucosal barrier of gastric mucosa.