

EVALUATION OF DUODENOGASTRIC  
REFLUX AFTER CHOLECYSTECTOMY

THESIS

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By

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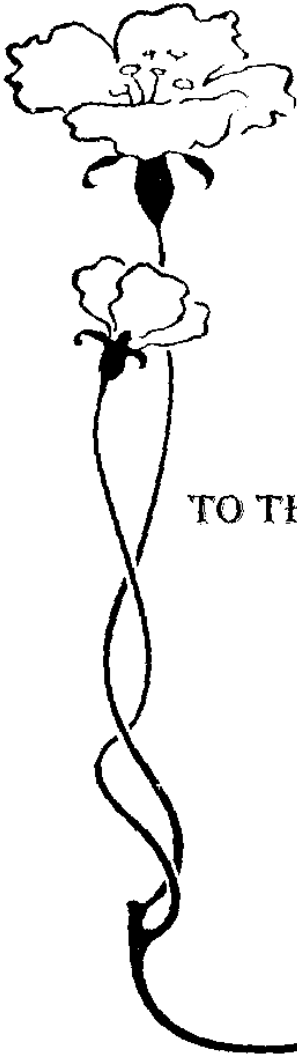
بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

﴿قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا إِنَّكَ  
أَنْتَ الْعَلِيمُ الْحَكِيمُ﴾

سورة البقرة

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TO THE GOOD SOULS OF MY  
PARENTS



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**INTRODUCTION  
AND AIM OF  
THE WORK**



## INTRODUCTION

Intermittent reflux of the duodenal contents into the stomach (duodenogastric reflux) is a physiological event, occurring both in the fasting state and after feeding. In the presence of normal acid secretion and normal gastric emptying, the potentially injurious refluxed duodenal contents are neutralized, diluted and cleared from the stomach (Wilson et al., 1993).

In the absence of the gall bladder function, as after cholecystectomy, and in cases with non functioning gall bladder, the constant flow of bile into the duodenum will result in excessive duodenogastric reflux (Buxbaum, 1982).

In the recent years several investigators have provided evidence that duodenogastric reflux is increased after cholecystectomy and that the increase in reflux may be greater in the symptomatic patient (Stahlberg et al., 1987). Some authors have reported an association between cholecystectomy and chronic antral superficial gastritis (Warshaw et al., 1979 and Kalima et al., 1981).

The effects of duodenogastric reflux on the gastric mucosa are controversial. Some investigators suggest that there may be a correlation between the amount of duodenogastric reflux and the mucosal damage. This hypothesis is also supported by the fact that chronic superficial gastritis is

*Introduction*

more frequent in the antrum, an area closure to the reflux (Larusso et al., 1990). The increase in the duodenogastric reflux in these patients may be due to the loss of the gall bladder as a bile reservoir, leading to a constant supply of bile into the duodenum, even when fasting (Moody et al., 1986 and Muller- Lisser et al., 1978)

The excessive entogastric reflux is the principle cause of a much wider constellation (group) of functional and morphological postoperative abnormalities, including persistent epigastric pain, nausea, bilious vomiting, weight loss, hypochlorohydrria and anaemia, all associated with severe endoscopic and histologic gastritis (Wallace et al., 1995).