

**ULTRASONOGRAPHY OF COLON  
USING WATER AS CONTRAST AGENT**

By  
**Kanu Gopal Bala**  
Doctor of Philosophy Student

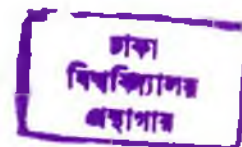
**This thesis is submitted in partial fulfillment of the requirements  
for the degree of Doctor of Philosophy**



400508

**Faculty of Postgraduate Medical Science and Research  
The Dhaka University**

2001



SHA

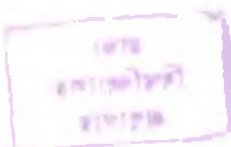
R

611.347

BAU

c.i

Se. Sec.



STATEMENT OF THE STUDENT

I do hereby declare that, examinations and interpretations of all the cases presented in the thesis entitled 'Ultrasonography of colon using water as contrast agent' are done by me and no part of it has been presented previously for a higher degree.

The research work was carried out in the Institute of Nuclear Medicine, Bangabandhu Sheikh Mujib Medical University, Shahbagh, Dhaka 1000 and in the Training & Research Centre of the Bangladesh Institute of Ultrasound in Medicine & Research, University of Science & Technology Chittagong, Eastern Plaza, Dhaka 1205 under the guidance of Professor Mirza Mazharul Islam, Professor of Surgery, Bangladesh Institute of Research and Rehabilitation of the Diabetic, Endocrine & Metabolic Disorders and Professor M. A. Karim, Director of the Institute of Nuclear Medicine, Bangabandhu Sheikh Mujib Medical University, Shahbagh, Dhaka 1000.

*Kanu Gopal Bala*

**Kanu Gopal Bala**  
Candidate

400508



CERTIFICATE OF THE SUPERVISORS

The works of the thesis entitled 'Ultrasonography of colon using water as contrast agent', for partial fulfillment of the requirements for the degree of 'Doctor of Philosophy' in Medical Science of Kanu Gopal Bala were done under our direct supervisions. He is sincere and hard working. The whole thesis is his own works. We are fully satisfied with his works.



**Prof. Mirza Mazharul Islam**

Professor of Surgery

Bangladesh Institute of Research and Rehabilitation  
of Diabetic, Endocrine & Metabolic Disorders  
Shahbagh, Dhaka 1000, Bangladesh



**Prof. M. A. Karim**

Director

Institute of Nuclear Medicine  
Bangabandhu Sheikh Mujib Medical University  
Shahbagh, Dhaka 1000, Bangladesh

400508



This thesis entitled 'Ultrasonography of colon using water as contrast agent' is submitted to the Faculty of Postgraduate Medical Science and Research, Dhaka University for the partial fulfillment of the requirements for the degree of Doctor of Philosophy on .14.JUNE. 2001.

Signature



**Kanu Gopal Bala**

Candidate



**Prof. Mirza Mazharul Islam**

Professor of Surgery

Bangladesh Institute of Research and Rehabilitation  
of Diabetic, Endocrine & Metabolic Disorders

Shahbagh, Dhaka 1000, Bangladesh

Supervisor



**Prof. M. A. Karim**

Director

Institute of Nuclear Medicine

Bangabandhu Sheikh Mujib Medical University

Shahbagh, Dhaka 1000, Bangladesh

Supervisor



**Prof. Syed Modasser Ali**

Dean

Faculty of Postgraduate Medical Science and Research

Dhaka University

Dean

This thesis entitled 'Ultrasonography of colon using water as contrast agent' is accepted by the Faculty of Postgraduate Medical Science and Research, Dhaka University for the partial fulfillment of the requirements for the degree of Doctor of Philosophy on ..... 2001.

**Convenor**

**Member**

**Member**

**Dean**

Faculty of Postgraduate Medical Science and Research  
Dhaka University

We further agree that, Kanu Gopal Bala has satisfactorily defended his thesis entitled 'Ultrasonography of colon using water as contrast agent' at the Examination of the Faculty of Postgraduate Medical Science and Research, Dhaka University for the degree of Doctor of Philosophy on ..... 2001.

**Convenor**

**Member**

**Member**

**Dean**

Faculty of Postgraduate Medical Science and Research  
Dhaka University

## ACKNOWLEDGEMENT

I would like to express my appreciation and deepest sense of gratitude and indebtedness to Professor Mirza Mazharul Islam, Professor of Surgery, Bangladesh Institute of Research and Rehabilitation of Diabetic Endocrine & Metabolic Disorders, Dhaka, Bangladesh and Professor M. A. Karim, Director, Institute of Nuclear Medicine, Bangabandhu Sheikh Mujib Medical University, Dhaka for suggesting this problem, their valuable advice, constant supervision, guidance, constructive criticism and critical reading of the manuscript without which it would have been impossible for me to complete this study.

I should like to express my appreciation and gratitude to Professor Syed Modasser Ali, Dean of Postgraduate Medical Science and Research, Dhaka University, Professor & Director of the National Institute of Ophthalmology and President & Chairman of the Bangladesh Medical Research Council for his invaluable help, cooperation, support and encouragement for completion of this study.



I am grateful to Professor Md. Suhrab Ali, Ex-Dean of the Postgraduate Medical Science and Research, Dhaka University for accepting me in the Ph.D. programme under Dhaka University.

I am sincerely thankful to Prof. M. A. Karim, Director of the Institute of Nuclear Medicine, Bangabandhu Sheikh Mujib Medical University, Shahbagh, Dhaka and Dr. Almas Begum, Chairman of the Training & Research Centre and Director of the Bangladesh Institute of Ultrasound in Medicine & Research, University of Science & Technology Chittagong, Eastern Plaza, Dhaka for kindly allowing and providing me with all the facilities available in their Institutes for examining and interpreting the patients for the study.

I would like to express my deep gratitude to National Professor N. Islam, Founder & Vice Chancellor of the University of Science & Technology Chittagong and Professor Soren Hancke, Secretary of the World Federation for the Ultrasound in Medicine & Biology and Professor of the University of Copenhagen for their deepest concern and encouragement for my work.

I am thankful to the authority of Mina Laboratory, Dhaka for allowing and providing me with all the facilities available for Double contrast barium enema X-ray, Colonoscopy and Histopathology necessary for the study.

I am thankful to all the patients for their active cooperation for being included in this study, without which it would have not been possible to carry out this study.

I am thankful to the Members of the Faculty of Postgraduate Medical Science and Research for allowing me to present this works in progress in two seminars, appreciating and encouraging me to complete the work.

For successful completion of this study I would also like to express my deepest gratitude to Professor Bernd Limberg, Department of Internal Medicine, Hospital of the University of Gottingen, Braunschweig, Germany, pioneer in the hydrocolonic sonography, for his expert suggestion and encouragement to complete the study.

I am very much thankful to Professor Barry B. Goldberg, Professor of Radiology and Director of Jefferson Ultrasound Research and Education

Institute, Thomas Jefferson University, Philadelphia, USA and Professor Hiroki Watanabe, Professor of Urology of Meiji University, Japan and President of the World Federation for Ultrasound in Medicine and Biology for their keen interest in my study.

I would like to express my appreciation to my well-wisher and friend Dr. Fauzia Moslem, Mr. M. H. Hohsin, for initiating and completing my work.

I am thankful to Mr. Humayun Kabir, Mr. Shah Alam, Mrs. Nazma Begum, Mr. Sharif and other staff of above mentioned Institutions and laboratory for their valuable cooperation.

Lastly, but not the least, my heartfelt appreciation and gratitude to my children Engr. Pabitra Bala, Nupur Bala and Poppy Bala for moral support, cooperation aspiration before and during this study.

Kanu Gopal Bala

Dhaka, Bangladesh

**TABLE OF CONTENTS**

TITLE PAGE	...	...	i
STATEMENTS	...	...	ii-iii
ACCEPTANCE PAGES	...	...	iv-vi
ACKNOWLEDGEMENTS	...	...	vii-x
TABLE OF CONTENTS	...	...	xi-xii
LIST OF FIGURES	...	...	xiii-xv
LIST OF TABLES	...	...	xvi
LIST OF CHARTS	...	...	xvii
LIST OF APPENDIXES	...	...	xviii
ABSTRACT	...	...	xix-xxiv
<b>Chapter:</b>			
I.	INTRODUCTION:	...	1-6
	I-1 Introduction	...	2
	I-2 Statement of the problem	...	3
	I-3 Purpose of the study	...	4
	I-4 Assumption	...	6
	I-5 Hypothesis	...	6
II.	REVIEW OF RELATED LITERATURES:	...	7-62
	II-1 Anatomic considerations	...	8
	II-2 Physiologic considerations	...	18
	II-3 Inflammatory bowel diseases	...	20
	II-4 Colon polyps and malignancies	...	35
	II-5 Clinical features of colon diseases	...	43
	II-6 Imaging studies of colon	...	47
	II-7 Ultrasonography of colon	...	52
III.	MATERIALS:	...	63-86
	III-1 Period and place of study	...	64
	III-2 Number of patients	...	65
	III-3 Water as contrast agents	...	71
	III-4 Ultrasound instruments	...	74
	III-5 Ultrasound image recording devices	...	80

III-6	Accessory devices	...	...	82
III-7	X-ray instrument	...	...	85
IV.	METHODS:	...	...	87-159
IV-1	Hydrocolonic sonography	...	...	87
IV-2	Hydrocolonosonographic diagnosis	...	...	92
IV-3	Conventional abdominal sonography	...	...	117
IV-4	In vitro ultrasound examination of colon	...	...	126
IV-5	Double contrast barium enema x-ray	...	...	131
IV-6	Histopathological diagnosis of colon diseases	...	...	145
IV-7	Method of calculation	...	...	159
V.	RESULTS:	...	...	160-167
V-1	Total patients	...	...	161
V-2	Group-I patients	...	...	163
V-3	Group-II patients	...	...	165
VI.	DISCUSSIONS:	...	...	168-178
VI-1	Discussion of the findings	...	...	169
VI-2	Confirmation with other studies	...	...	174
VI-3	Limitations	...	...	177
VI-4	Recommendations	...	...	178
VII.	CONCLUSIONS:	...	...	179-183
VII-1	Summary	...	...	180
VII-2	Findings	...	...	181
VII-3	Conclusions	...	...	182
VII-3	Implementations	...	...	183
VII-5	Recommendations for further study	...	...	183
VIII.	REFERENCES:	...	...	184-198
IX.	APPENDIXES:	...	...	199-205

## **LIST OF FIGURES**

	<u>Page No.</u>
Figure – II-1 : Developing alimentary canal	: 9
Figure – II-2 : Different parts of colon	: 11
Figure – II-3 : Histologic layers of colon	: 12
Figure – II-4 : Anatomy of colon wall	: 15
Figure – II-5 : Arterial blood supply of colon	: 16
Figure – II-6 : Diagram of sonography of colon	: 54
Figure – III-1 : Ultrasound contrast effect of water	: 73
Figure – III-2 : Aloka SSD-500 scanner	: 77
Figure – III-3 : Contron Sigma 21 scanner	: 78
Figure – III-4 : Aloka SSD-1100 scanner	: 79
Figure – III-5 : Video graphic ultrasound printer	: 81
Figure – III-6 : Conventional enema can	: 83
Figure – III-7 : Can & stand for instilling water	: 84
Figure – III-8 : X-ray unit	: 86
Figure – IV-1 : Water instillation through anus	: 91
Figure – IV-2 : Small amount of fecal material in colon	: 97
Figure – IV-3 : Colon with haustra	: 98

Figure – IV-4	: Normal descending colon	: 99
Figure – IV-5	: Normal transverse colon	: 100
Figure – IV-6	: Normal ascending colon	: 101
Figure – IV-7	: Normal caecum, Bauhin's valve, ileum	: 102
Figure – IV-8	: Colon wall in 5.0 & 7.5 MHz examination	: 103
Figure – IV-9	: Sonographic 5 layers of colon wall	: 104
Figure – IV-10	: 5 layers of intestine in endoluminal exam.	: 105
Figure – IV-11	: Small colon polyp	: 106
Figure – IV-12	: Large colon polyp	: 107
Figure – IV-13	: Villous adenoma	: 108
Figure – IV-14	: Carcinoma of colon	: 109
Figure – IV-15	: Carcinoma of colon-annular lesion	: 110
Figure – IV-16	: Carcinoma at rectosigmoid colon	: 111
Figure – IV-17	: Crohn's disease	: 112
Figure – IV-18	: Severe Crohn's disease	: 113
Figure – IV-19	: Ulcerative colitis	: 114
Figure – IV-20	: Pseudopolyposis in ulcerative colitis	: 115
Figure – IV-21	: Diagrammatic comparison of CD & UC	: 116
Figure – IV-22	: Normal bowel with target configuration	: 121
Figure – IV-23	: High frequency examination of antral wall	: 122

Figure – IV-24	: 5 layers of GIT in conventional USG	: 123
Figure – IV-25	: Thickening of bowel wall	: 124
Figure – IV-26	: Transabdominal ultrasound of ca-colon	: 125
Figure – IV-27	: Specimen of colon	: 128
Figure – IV-28	: USG examination of colon specimen	: 129
Figure – IV-29	: USG image of colon specimen	: 130
Figure – IV-30	: Radiological anatomy of normal colon	: 137
Figure – IV-31	: Barium enema x-ray of ileum & appendix	: 138
Figure – IV-32	: BEX of early Crohn's disease	: 139
Figure – IV-33	: BEX of benign sessile tumour of colon	: 140
Figure – IV-34	: BEX of familial polyposis	: 141
Figure – IV-35	: BEX of rectosigmoid carcinoma	: 142
Figure – IV-36	: BEX of annular carcinoma of colon	: 143
Figure – IV-37	: Barium enema spot films	: 144
Figure – IV-38	: Histopathology of normal colon	: 154
Figure – IV-39	: Histopathology of Crohn's disease	: 155
Figure – IV-40	: Histopathology of tuberculosis of colon	: 156
Figure – IV-41	: Histopathology of colon polyp	: 157
Figure – IV-42	: Histopathology of carcinoma of colon	: 158



## LIST OF TABLES

Table – V-1	: Comparison of HS and BEX diagnosis of colon diseases ... ..	: 164
Table – V-2	: Colonoscopic and or surgical diagnosis & Comparison with HS & BEX ... ..	: 166
Table – V-3	: Nosologic sensitivity & specificity, Diagnostic sensitivity & specificity ...	: 167

**LIST OF CHARTS**

Chart – III-1	: Age & sex distributions of the total patients included into the study	...	: 68
Chart – III-2	: Age & sex distributions of the Group-I patients	... ..	: 69
Chart – III-3	: Age & sex distributions of the Group-II patients	... ..	: 70
Chart – V-1	: Comparison of patients having complete examination with incomplete exam.	...	: 162

## **LIST OF APPENDIXES**

Appendix – IX-A	: History sheet and consent of patient	: 200
Appendix – IX-B	: HS findings and impressions	: 201
Appendix – IX-C	: HS report page-1	: 202
Appendix – IX-D	: HS report page-2	: 203
Appendix – IX-E	: Prof. Bernd Limberg letter no. 1	: 204
Appendix – IX-F	: Prof. Bernd Limberg letter no. 2	: 205

## ABSTRACT

---

**ULTRASONOGRAPHY OF COLON**  
**USING WATER AS CONTRAST AGENT**

[Thesis Abstract]  
Kanu Gopal Bala  
Dhaka University

**PROBLEM**

The problem of this study was to investigate whether after instilling water into colon through anus, colon can be examined thoroughly and efficiently by ultrasonography. The usefulness of the procedure is compared with that of double contrast barium enema x-ray.

This is an applied research. The purpose of this study is to find out a workable procedure of examining colon by ultrasonography. If the procedure is proved equally or more efficient than that of double contrast barium enema x-ray for diagnosis of colon pathologies, the procedure can be applied for routine examining procedure. It is assumed that, after proper colon preparation to make it gas and stool free, if water is instilled into colon through anus the colon will be distended and is easily identified by par abdominal ultrasound examination. Then identification of normal colon wall and colon pathologies will be possible.

## **PROCEDURES**

During a 23month's period from July 1999 to May 2001 patients were a total 523 patients were examined. 15 patients were dropped from the study due to inadequate preparation and the patients refused take further preparations. Finally 508 patients were included into the study.

Out of the 508 patients, 457 [89.96%] were male and 51 [10.04%] were female. Age ranged from 14 to 78 years, mean being 48.73 years. A history sheet was filled up by the information obtained from each of the patients. The study protocol was approved by the Faculty of Postgraduate Medical Science & Research of Dhaka University. All the 508 patients included into the study were subdivided into Group-I and Group-II. Total 368 patients were included into Group-I. All the patients were examined by double contrast barium x-ray with the same preparations in the same day. If the examination was deferred to a next day, a separate preparation was given. Total 140 patients were included into Group-II. All the patients were examined by double contrast barium x-ray. All the patients were examined either by colonoscopy and biopsy and or surgery.

Patients were instructed to eat an exclusively low-residue diet during the previous day of examination. On the next morning, after bowel moved, the

patient came to the centre in empty stomach. At the centre, enema simplex with warm soap-water was given. Usually 1-2 enemas were sufficient to clean the entire colon. No significant adverse effects were noticed during or after colon cleaning.

A total up to 1500 ml of water was instilled into colon through anus using an specially prepared big sized enema can and a stand. Continuous abdominal sonographic examination of the colon from the time of water instillation was carried out with real-time scanning device. No buscopan or any spasmolytics were used.

Examination began as soon as the water began to instill into colon through anus. Rolling or tilting of patient were not necessary. The examination began at rectosigmoid area and gradually covered descending colon, splenic flexure, transverse colon, hepatic flexure, ascending colon, caecum and terminal part of colon. The colon lumen, contents, wall and surrounding areas were evaluated. Usually 15 to 20 minutes were sufficient to complete the examinations. Presence of small amount of faeces did not interfere with real-time examination of the colon and easily could be differentiated from colon mass.

## **FINDINGS**

Out of 523 patients, in 508 [97.13%] patients it was possible to evaluate the entire length of colon starting at the rectosigmoid junction and ending at the caecum by hydrocolonic sonography. In the 15 [2.87%] patients, the examination could not be done completely due to presence of gas in the colon and the patients refused to take further enema for completing the examination. The procedure was well tolerated by each of the patients. There was no need to give hyasomide injection to the patients for relaxation of the bowel. Total examination time was 15-20 minutes.

The patients were classified into Group-I and Group-II according to confirmation of the final diagnosis by colonoscopy and or surgery. Total 368 patients were included into group-I. The patients had HS & BEX examinations. Diagnoses were made based on the sonographic and radiographic findings of the examinations respectively. Out of 368 patients, hydrocolonic sonography [HS] detected 244 [66.30%] patients as normal with all the 5 layers of wall and normal haustra. 38 [10.33%] had carcinoma, 35 [9.51%] polyp, 20 [5.43%] Crohn's disease, 27 [7.34%] ulcerative colitis and 4 [1.09%] had diverticulitis.



Total 140 patients were included into group-II. After proper preparation all the patients were examined by hydrocolonic sonography. All the patients were examined by BEX, colonoscopy and or surgery. results were presented

Nosologic sensitivity, nosologic specificity, diagnostic sensitivity and diagnostic specificity of HS and BEX were calculated against the diagnosis done by colonoscopy and or surgery. Predictive value of negative HS & BEX and predictive value of positive HS & BEX were also calculated accordingly.

### **CONCLUSIONS**

Hydrocolonic sonography is a significant useful diagnostic imaging modality to evaluate colon diseases. Diagnostic sensitivity and specificity of HS are comparable, even better, in comparison to double contrast barium enema x-ray. The procedure is well tolerated and acceptable to the patients. No significant adverse effect is noticed during or after the examination. There is no ionizing radiation for the ultrasound examination. The modality is cheap and without any adverse effect. The examination of colon by HS can be introduced in the country without any significant involvement of extra money or man-power.

# INTRODUCTION

---

## CHAPTER 1

## **I: INTRODUCTION**

### **I-1: INTRODUCTION:**

The colon, approximately 3 to 5 feet in length, extends from ileum to rectum. The colon is much more than a receptacle and conduit for the end products of digestion. A variety of diseases affect colon i.e, disturbances of colon physiology, infections, inflammatory bowel diseases and neoplastic diseases. Different types of diagnostic modalities are used to evaluate the colon pathologies. Colonoscopic examination can see inner surface of colon directly. But it is too much invasive and pathologies deep to mucosa can be missed easily. Conventional single contrast and double-contrast barium enema x-ray [BEX] of colon can delineate the lumen of colon and has become standard method of examination. Radionuclide scanning, CT scanning & MRI have a relatively restricted application. These modalities are not widely available and not cost-effective in our country.

Diverticular disease of colon are common in middle-aged and elderly subjects. The incidence in western communities is between 5 and 10% in those over age of 60. Chron's disease and ulcerative colitis are non-specific inflammatory conditions of colon affecting 5 - 10 and 2-6 individuals per 100000. The incidence of neoplastic polyps closely parallels that of colonic cancer. In western countries, neoplastic polyps occur in up to 10% of the population over the age of 40, whereas incidence of colon cancer approaches 60 per 100000. So, colon diseases cause considerable morbidity & mortality, especially in middle-aged and elderly age group. There is thus, an alternative diagnostic modality is needed for colon pathologies.

#### **I-2: STATEMENT OF THE PROBLEM:**

Ultrasonography has been tried to evaluate colon with a limited success. The major part of colon cannot be consistently identified. The collapsed transverse colon may be located below the pancreas and stomach from its gas content. Pseudomasses may be caused by fluid at splenic flexures. The sigmoid colon and rectum may also cause pseudomasses in the pelvis, and use of water enema is advocated for differentiation.

In a number of pathological conditions the colon wall is thickened. Thickened bowel wall with central highly reflective complex area mimicking kidney image. This is termed "Pseudokidney" sign and, though non-specific, strongly suggests bowel pathology. Ultrasound examination of gastrointestinal tract may be difficult to image in most patients without special preparations. The gastrointestinal tracts containing water or some other acoustic transmittable contrast agent can be delineated easily with ultrasound. Many laboratories have begun to investigate various contrast agents in pursuit of the ideal medium for imaging the stomach, duodenum, small bowel and colon.

The problem of this study was to investigate whether after instilling water into colon through anus, colon can be examined thoroughly and efficiently by ultrasonography.

### **I-3: PURPOSE OF THE STUDY:**

The purpose of this study is to find out a workable procedure of examining colon by ultrasonography. If the procedure is proved equally or more efficient than that of double contrast barium enema x-ray for diagnosis of

colon pathologies, the procedure can be applied for routine examining procedure.

In normal conditions colon contains fecal materials and gases. Both interfere with thorough sound transmission. Fecal materials can be eliminated from the colon by giving colon preparations. Instilling water inside colon through anus eliminates gases from colon, distends and delineates colon wall. Water acts as negative contrast and allows examining side walls and distal-walls of colon.

Because water is an ideal ultrasonic contrast medium, the instillation of water into the colon would improve the diagnostic value of ultrasonography in evaluating inflammatory or neoplastic diseases involving the colon. By instilling water into colon, it is possible to demonstrate the entire colon starting at the recto-sigmoidal boundary and ending at the caecum. Aside from demonstrating the intestinal lumen, a high-resolution transducer will also allow detailed evaluation of the intestinal wall and its surrounding tissue.

**I-4: ASSUMPTION:**

The type of research is applied research. It is assumed that, after proper colon preparation to make it gas and stool free, if water is instilled into colon through anus the colon will be distended and is easily identified by par abdominal ultrasound examination. Then identification of normal colon wall and colon pathologies will be possible.

**I-5: HYPOTHESIS:**

Colon can be examined par abdominal ultrasonography after instilling water into colon through anus. This procedure is comparable or even better than that of double contrast barium enema x-ray.

**REVIEW OF LITERATURES**  
**CHAPTER 2**



## II. REVIEW OF LITERATURES

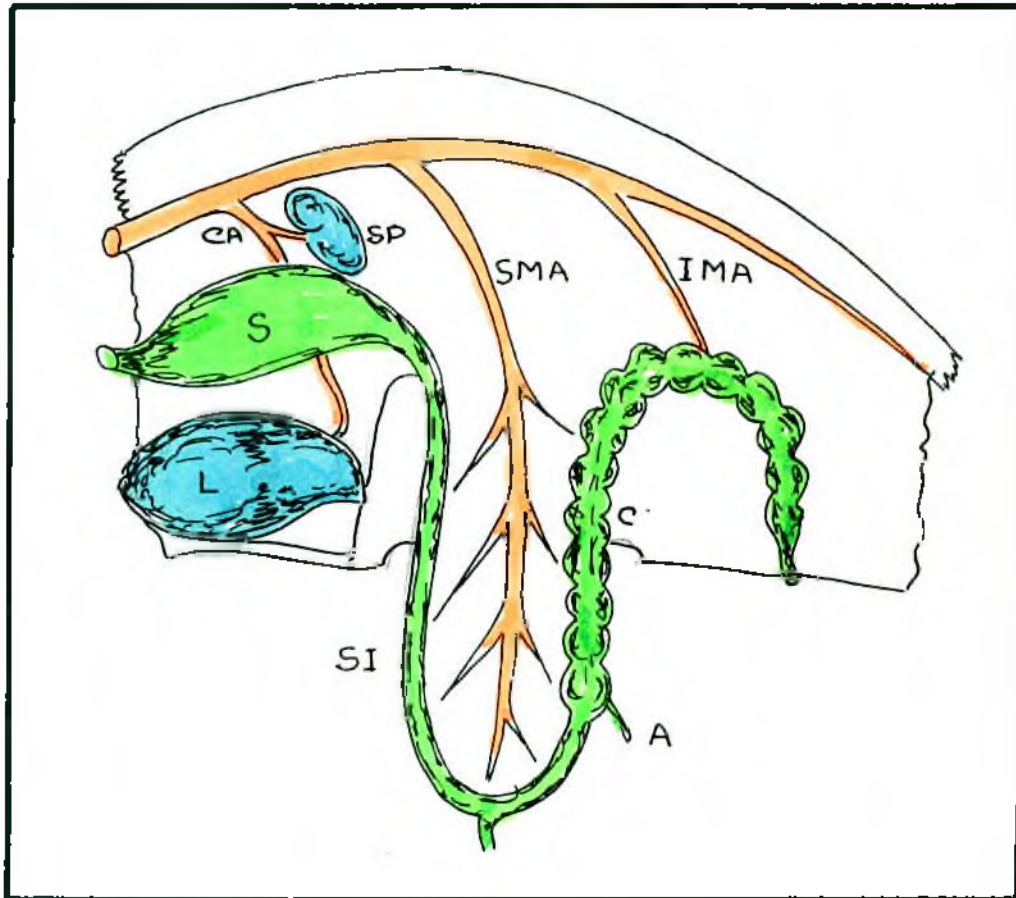
### **II-1: ANATOMIC CONSIDERATION :**

#### **Embryology**

The primitive gut, which is divided into the foregut, the midgut, and the hindgut, develops during the fourth week of pregnancy. The midgut develops into the small intestine and the large intestine proximal to the mid-transverse colon. This intestinal segment receives blood from the superior mesenteric artery. The hindgut develops into the large bowel distal to the mid-transverse colon as well as the proximal anus and the lower urogenital tract, and receives its blood supply from the inferior mesenteric artery.

The developing midgut migrates out of the abdominal cavity during the sixth week of pregnancy. During the ensuing 4 weeks, the midgut rotates  $270^{\circ}$  in a counterclockwise direction about the superior mesenteric artery before assuming its final anatomic position in the abdominal cavity [Fig – II-1].

Figure No. – II-1



Representation of the developing alimentary canal, viewed from the left, at the stage of herniation of the mid-gut. S – Stomach, SI – Small intestine, A – Appendix, C – Colon, L – Liver, SP – Spleen, CA – Celiac artery, SMC – Superior mesenteric artery, IMA – Inferior mesenteric artery.

## **Anatomy**

The colon, approximately 3 to 5 feet in length, extends from the ileum to the rectum [Fig – II-2]. The terminal ileum joins the caecum on its posteromedial border at the ileocaecal valve. The fold of Treves is located on the distal ileum just proximal to the ileocaecal valve. The caecum projects from the antimesenteric of the ascending colon and is a large blind pouch with no mesentery. The caecum is approximately 7.5 to 8.5 cm in diameter and is the widest portion of the colon. The colon progressively diminishes in size to the sigmoid colon, its narrowest portion, which is approximately 2.5cm in diameter.

The layers of the colon wall include mucosa, submucosa, inner circular muscle, outer longitudinal muscle, and serosa [Fig – II-3]. The longitudinal muscle is separated into three distinct bands called teniae coli positioned  $120^{\circ}$  apart about the circumference of the colon. The teniae converge proximally at the appendix and disappear as distinct bands at the proximal rectum at the level of the sacral promontory. Haustra coli are sacculations between the teniae and are separated by crescent-shaped folds called plicae semilunares. Appendices epiploicae are fatty appendages attached to teniae.

The omentum is attached to the transverse colon on its anterior superior edge. The ascending colon, descending colon, and posterior surface of the

Figure No. – II-2

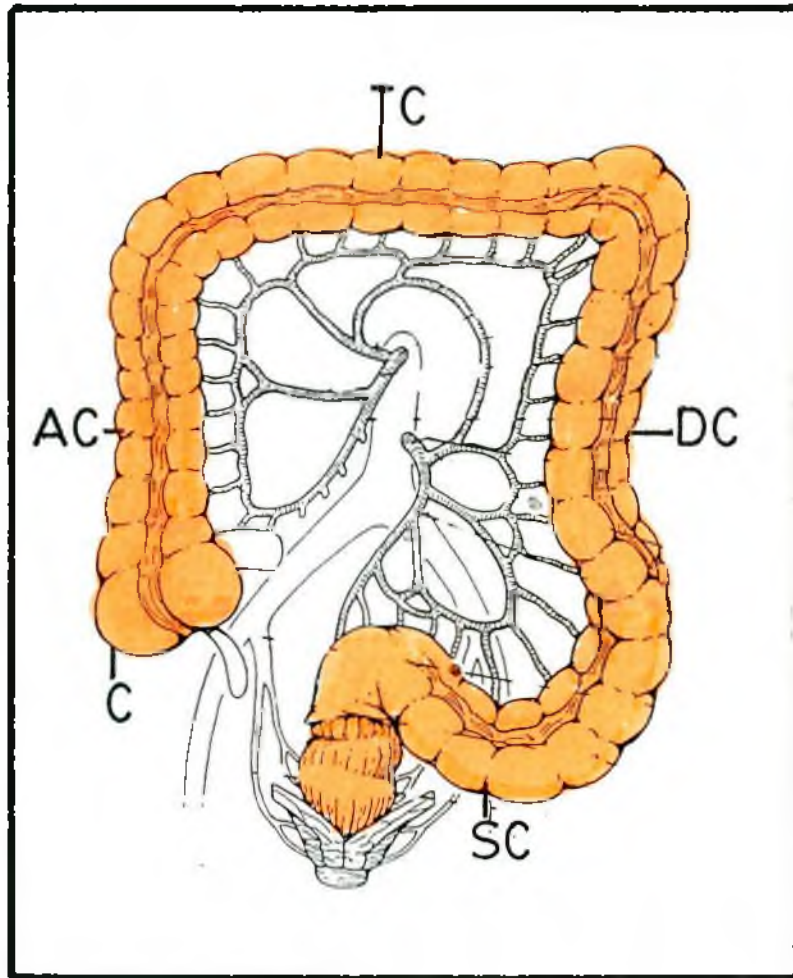
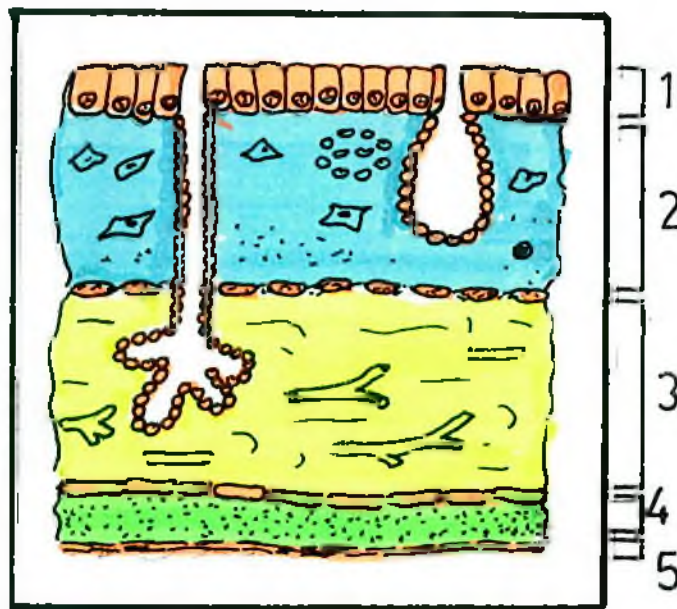


Diagram showing different parts of colon. C – Caecum, AC – Ascending colon, TC – Transverse colon, DC – Descending colon, SC – Sigmoid colon.

Figure No. – II-3



Schematic representation of histologic layers of colon which correspond with the sonographic layering. 1 – Interface of superficial mucosa with luminal content, 2 – Deeper mucosa including muscularis mucosa, 3 – Submucosa, 4 – Muscularis propria, 5 – Serosa.

hepatic and splenic flexures are usually retroperitoneal, whereas the caecum, transverse colon, and sigmoid colon are intraperitoneal in location.

**Histology:**

The large intestine consists of a mucosal membrane with no folds except in its distal [rectal] portion. No villi are present in this portion of the intestine. The intestinal glands are long and characterized a great abundance of goblet and absorptive cells and a small number of enteroendocrine cells. The absorptive cells are columnar and have short, irregular microvilli. The large intestine is well suited to its main functions: absorption of water, formation of the faecal mass, and production of mucus. Mucus is a highly hydrated gel that not only lubricates the intestinal surface but also covers bacteria and particulate matter. The absorption of water is passive, following the active transport of sodium out of the basal surfaces of the epithelial cells.

The lamina propria is rich in lymphoid cells and in nodules that frequently extend into the submucosa. The richness in lymphoid tissue is related to abundant bacterial population of the large intestine. The muscularis comprises longitudinal and circular strands. This layer differs from that of the small intestine, because fibres of the outer longitudinal layer congregate in three thick bands called tenia coli. In the intraperitoneal portions of the

colon, the serous layer is characterized by small, pendulous protuberances composed of adipose tissue – the appendices epiploicae [Fig – II-4].

### **Arterial Supply.**

The superior mesenteric artery arises from the ventral surface of the aorta just below the celiac axis. It passes downward behind the pancreas and crosses in front of the third portion of the duodenum. It supplies the cecum, ascending colon, and transverse colon via its ileocolic, right colic, and middle colic branches [Fig – II-5].

### **Venous Drainage:**

Except for the inferior mesenteric vein which lies adjacent to the ascending branch of the left colic artery, the veins draining the colon, and proximal rectum. It runs in a retroperitoneal location to the left of the ligament of Treitz, continues behind the body of the pancreas, and enters the splenic vein. The superior mesenteric vein drains the caecum, ascending colon, and transverse colon and joins the splenic vein to form the portal vein.

### **Lymphatic Drainage:**

The colon is encircled by lymphatic channels located in the submucosa and the muscularis mucosae. The mucosa has rich vascular plexi but no lymphatics. Lymphatic vessels follow the arterial supply of the colon. Lymph nodes are located on the bowel wall, along the inner margin of the

Figure No. – II-4

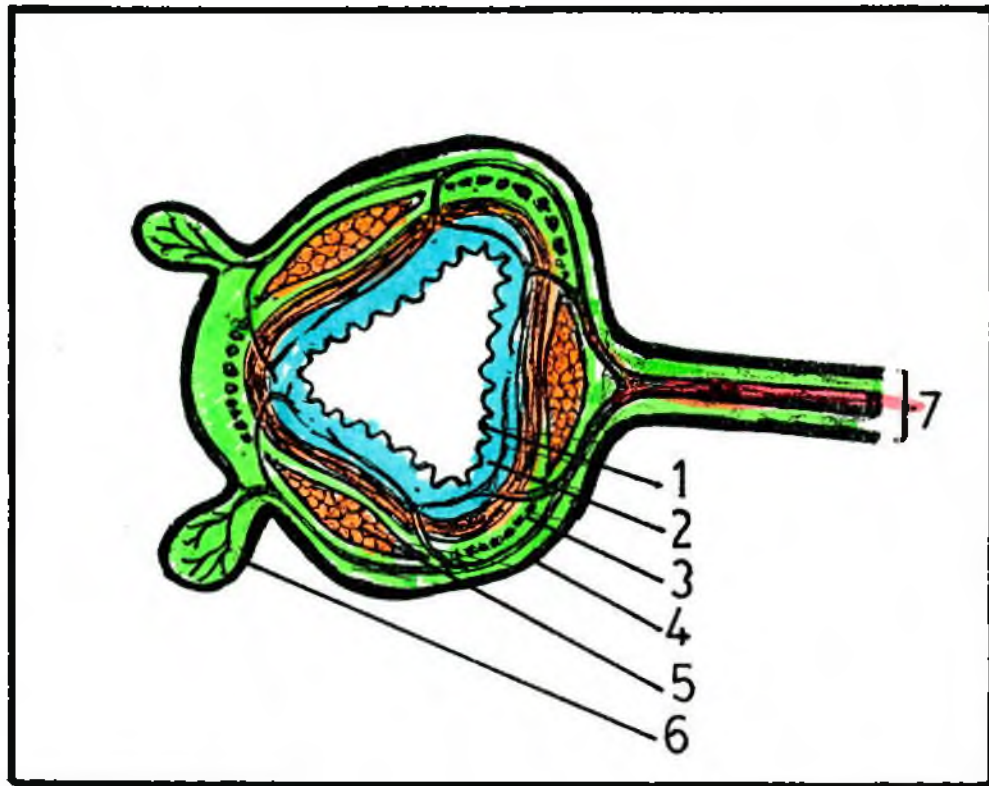
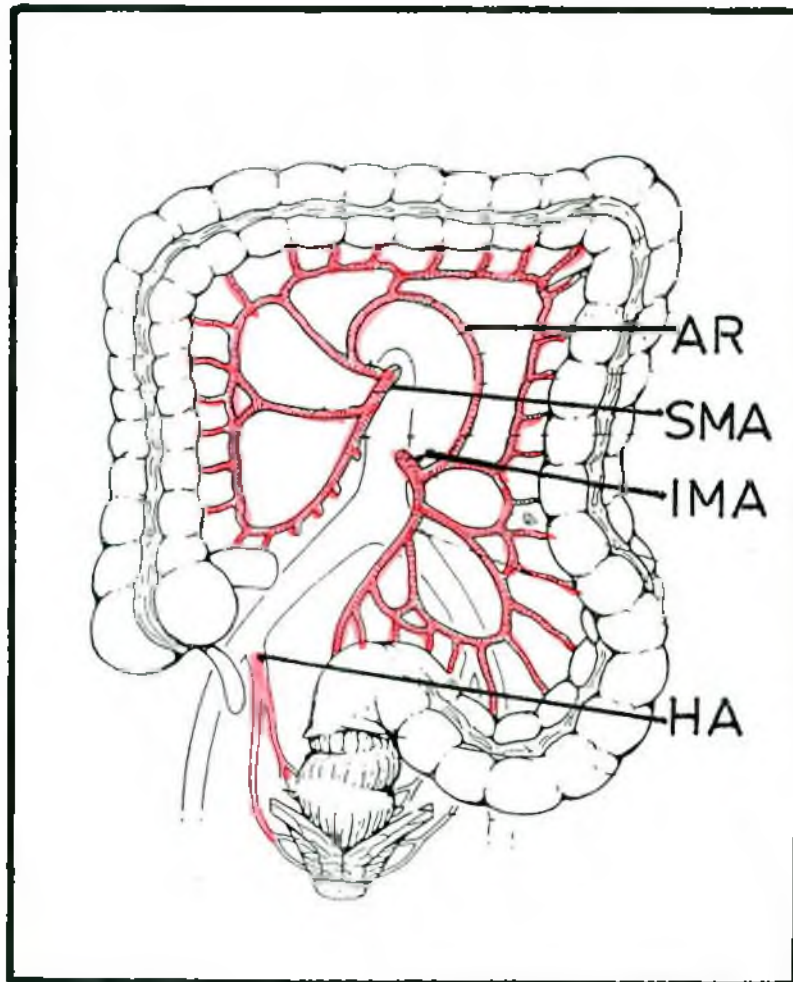


Diagram showing anatomy of colonic wall. 1 – Mucosa, 2 – Submucosa, 3 – Muscularis, 4 – Serosa, 5 – Tenia [modified longitudinal muscle], 6 – Appendix epiploica, 7 – Mesocolon with intestinal arcade.



Figure No. – II-5



Arterial blood supply of colon and rectum. SMA – Superior mesenteric artery, IMA – Inferior mesenteric artery, HA – Hypogastric artery, AR – Arc of Riolan.

bowel, around the named mesenteric arteries, and about the origin of the superior and inferior arteries.

**Nerve Supply:**

Sympathetic nerves inhibit and parasympathetic nerves stimulate peristalsis.

## **II-2: PHYSIOLOGIC CONSIDERATION:**

### **Normal Colonic Function:**

The colon is much more than a receptacle and conduit for the end products of digestion. This organ absorbs water, sodium, and chloride, and secretes potassium, bicarbonate, and mucus; it is the site of digestion of certain carbohydrates and proteins, and provides the environment for the bacterial production of vitamin K.

### **Water and Electrolyte Exchanges:**

The colon receives from 900 to 1500 ml of ileal fluid each day, of which all but 100 to 200 ml is absorbed.

### **Sodium and Potassium:**

Sodium is absorbed against concentration and electrical gradients under the influence of sodium pump. Potassium transport is passive; potassium molecules move along the gradient generated by active sodium transport. The energy for this sodium-potassium exchange appears to be provided by the metabolism of short-chain fatty acids.

### **Chloride and Bicarbonate:**

Chloride, like sodium, is actively absorbed across the colonic mucosa against a concentration gradient. Chloride and bicarbonate are exchanged at

the luminal border. Chloride absorption is facilitated by an acidotic environment, and the secretion of bicarbonate is enhanced by increased concentration of luminal chloride.

**Ammonia:**

Colonic bacteria degrade protein and urea to produce ammonia. Ammonium ions react with bicarbonate to form nonionized ammonia and carbon dioxide. The nonionized ammonia diffuses across the colonic mucosa and is carried to the liver through the portal vein. Ammonia absorption becomes a problem in patients with severe liver disease, who are unable to metabolize ammonium.

### **II-3: INFLAMMATORY BOWEL DISEASES**

#### **Historical review:**

Until recently it has been fashionable to attribute the first 'modern' description of ulcerative colitis to Sir Samuel Wilks who, in a letter to the *Medical Times and Gazette*, referred to a death from idiopathic dysentery and suggested that the ulcerated bowel was caused by an abortifacient (Wilks 1859). It was Wilks again, in lectures on pathological anatomy, who first made the distinction between ulcerative colitis and specific forms of dysentery (Wilks & Moxon 1875).

Wilks and Moxon describe a severe, highly vascular inflammation of the entire colon with scattered minute points of ulceration in a young woman who died after experiencing severe bloody and mucoid diarrhea.

Habershon, writing in *Diseases of the Abdomen*, published in London in 1862, was probably the first to describe intestinal pseudopolyps in ulcerative colitis (Habershon 1862).

At the Paris Congress of Medicine in 1913 there was further discussion on ulcerative colitis, although it was largely a regurgitation of the ideas expressed 4 years earlier in London.

Although the workers from the Mount Sinai Hospital in New York put the disease that we now call Crohn's disease on the medical map, others had clearly recognized its occurrence as a distinct disease entity for at least a century before the most quoted paper of Crohn and colleagues (Crohn et al 1932). Historical surveys have revealed descriptions of apparent cases of Crohn's disease as far back in 1769 (Morgagni 1769). A clinical history and autopsy finding in a young man who died of intestinal obstruction after many years of abdominal pain were reported; the distal ileum was described as thickened and narrowed (Coombe 1813).

#### **Molecular Implications of Genetic Studies:**

Familial (Satsangi et al 1994) and twin (Tysk et al 1988) studies of the inheritance of Crohn's disease and ulcerative colitis have provided powerful evidence of a genetic contribution to IBD (inflammatory bowel disease). Identification of the genes that confer susceptibility to ulcerative colitis and Crohn's disease and study of the regulation of genes involved in their pathogenesis will undoubtedly provide powerful tools for diagnosis, insights into etiology and pathobiology, and identify potential targets for therapy.

Genetic studies of IBD have revealed significant heritable components to Crohn's disease and ulcerative colitis. Candidate genes have been investigated, and so far the strongest associations have been between HLA

alleles and IBD, particularly ulcerative colitis. These studies have clarified the distinction between Crohn's disease and ulcerative colitis, and suggest that there is heterogeneity within Crohn's disease and ulcerative colitis. Already genetic analyses of IBD have improved the accuracy of disease taxonomy, and may in future provide an adjunct to differential diagnosis. These disease associations described so far are open to a number of interpretations. Association with HLA suggests a role for these molecules in antigen presentation or T-cell selection in the pathobiology of IBD.

**Genetics of inflammatory bowel disease:**

Familial aggregation of inflammatory bowel disease has long been recognized. First-degree relatives (offspring siblings and parents) have the greater risk, particularly the siblings. The first-degree relatives of patients with ulcerative colitis or Crohn's disease have a 10-15 fold increase in the risk of having the same disease as the patient (Monsén et al 1987, Orholm et al 1991). However, the two diseases may also occur in the same family. This discordance of proband/relative pairs was 26% in a recent study (Reed et al 1992).

The data derived from studies in twins add strong support to the view that these disorders have a genetic component, since there is a significant increase in the concordance of inflammatory bowel disease in monozygotic

twins compared to dizygotic twins. In a Swedish study (Tysk et al 1988) eight of 18 monozygotic and only one of 26 dizygotic twin pairs were concordance for Crohn's disease.

Several genetic syndromes have been reported in association with inflammatory bowel disease. However, in only three has the association been consistent and therefore of possible pathogenic importance. These are Turner's syndrome, glycogen storage disease type Ib, and Hermansky-Pudlak syndrome.

Genes coded by chromosomes 2, 6 and 7 appear to be important for the susceptibility to suffer from inflammatory bowel disease. Even though not all these associations have been confirmed, interesting results have been published and are worth pursuing.

#### **Molecular Implication of Genetic Studies:**

Familial (Satsangi et al 1994) and twin (Tysk et al 1988) studies of the inheritance of Crohn's disease and ulcerative colitis have provided powerful evidence of a genetic contribution to IBD (inflammatory bowel disease). Identification of the genes that confer susceptibility to ulcerative colitis and Crohn's disease and study of the regulation of genes involved in their pathogenesis will undoubtedly provide powerful tools for diagnosis, insights into etiology and pathobiology, and identify potential targets for therapy.



Genetic studies of IBD have revealed significant heritable components to Crohn's disease and ulcerative colitis. Candidate genes have been investigated, and so far the strongest associations have been between HLA alleles and IBD, particularly ulcerative colitis. These studies have clarified the distinction between Crohn's disease and ulcerative colitis, and suggest that there is heterogeneity within Crohn's disease and ulcerative colitis. Already genetic analyses of IBD have improved the accuracy of disease taxonomy, and may in future provide an adjunct to differential diagnosis. These disease associations described so far are open to a number of interpretations. Association with HLA suggests a role for these molecules in antigen presentation or T-cell selection in the pathobiology

#### **Epidemiological Overview of inflammatory Bowel Disease:**

The study of the epidemiology of inflammatory bowel disease serves three purposes. It allows first, the measurement of the size of the problem that inflammatory bowel disease presents in the community, and secondly the way in which it is changing in frequency, or is associated with specific environmental features. Thirdly, it may provide clues to the etiology of the disease, which is poorly understood.

The substantial differences recorded in the frequencies of Crohn's disease and ulcerative colitis remain poorly understood. However, there are

indications that Crohn's disease becomes prominent if early life is in an urbanized community, whereas colitis may be more even in its impact, whether life is spent in urban or rural communities. There are also likely substantial differences in disease occurrence in European, particularly north European, populations or those with north European lifestyles and those living in tropical or subtropical areas, but clear confirmatory evidence is lacking, notably because endemic infectious disease make the confident attribution of non-specific disease difficult.

#### **Epidemiology of Inflammatory Bowel Disease:**

Whether antibodies against dietary antigens play a primary role in inflammatory bowel disease etiology or are secondary to inflammation is not known. There has, however, been a report of a potential relationship between cow's milk sensitivity during infancy and the subsequent development of ulcerative colitis (Galssman et al 1990).

Although found a significant deficiency of breast-feeding in patients with ulcerative colitis compared with controls was found, no such effect was seen in Crohn's disease (Whorwell et al 1979). In contrast, it was reported a significantly shorter breast-feeding period among Crohn's disease patients than in controls (Bergstrand and Hellers 1983).

Considerable interest has been raised by studies on the consumption of refined sugar in Crohn's disease. The intake of refined sugar was approximately double that of controls (Martini and Brandes 1976).

Several studies reported a decreased consumption of fruit or vegetables among patients with Crohn's disease (Kasper & Sommer 1979, Thornton et al 1979, Bianchi Porro & Panza 1985) and those with ulcerative colitis (Bianchi Porro & Panza 1985, Brandes et al 1979). Whereas a decreased consumption of total dietary fiber in patients with Crohn's disease was noticed.

Several authors have speculated that toothpaste may produce a potentially damaging effect to the gut (Chess et al 1950, Sullivan 1990).

#### **Epidemiology: Smoking and Oral Contraception:**

The association of non-smoking with ulcerative colitis was detected quiet earlier (Samuelsson 1976, Harries et al 1982). Crohn's disease patients tended to be smokers (Somerville et al 1984).

The suggestion that the development of inflammatory bowel disease might be related to oral contraception arose from case reports of women developing colitis shortly after starting oral contraceptives and improving after stopping (Kilpatrick et al 1968, Bonfils et al 1977, Heron et al 1981,

Tedesco et al 1982). Oral contraception is clearly associated with a small increase in risk of both Crohn's disease and ulcerative colitis.

### **Lymphocytes in Inflammatory Bowel Diseases:**

Lymphocytes are a key component of the cellular immune system, and play a major role in amplifying and maintaining the chronic mucosal inflammation seen in inflammatory bowel disease (IBD). In investigating the role of lymphocytes in IBD it is useful to consider the immune system as having separate compartments: systemic – represented by peripheral blood lymphocytes and intraepithelial lymphocytes.

### **Humoral Immunity in Inflammatory Bowel Diseases:**

Advances in the field of immunology have changed the way we view syndromes of autoimmunity, including inflammatory bowel diseases. Investigators have sought the presence of autoantibodies in inflammatory bowel disease both to explain the pathogenesis of the disease and to identify markers to distinguish idiopathic inflammatory bowel disease from other conditions. As early as the 1950s, scientists were looking for (and finding) colonic antibodies to explain the inflammation seen in ulcerative colitis (Broberger & Perlmann 1959, Lagercrantz et al 1966, Marcussen 1976, Das et al 1978, Snook et al 1991). Anticolon antibodies are also present in Crohn's disease but are less common. As with most autoantibodies, there is

evidence for and against anticolon antibodies having a pathogenic role in inflammatory bowel disease.

**Role of neutrophils in the pathogenesis of inflammatory bowel disease:**

Neutrophilic polymorphonuclear leukocytes (neutrophils) are the most prevalent leukocytes in the circulation, comprising approximately 70 – 80% of the total intravascular pool of leukocytes. The primary function of the neutrophil is to engulf and destroy invading microorganisms. In order to accomplish this task these cells have acquired the ability to synthesize and release large amounts of toxic reactive oxygen metabolites and proteinases. Although this inflammatory response is essential for maintaining normal health, excessive and/or inadvertent recruitment and metabolic activation of neutrophils may result in substantial injury to surrounding tissue (Klebanoff 1992). For example, it is well known that active episodes of inflammatory bowel disease (IBD) are characterized histologically by the extravasation and infiltration of large numbers of neutrophils. This enhanced inflammatory infiltrate is accompanied by extensive mucosal and/or transmural injury, including edema, crypt abscesses, loss of goblet cells, decreased mucus production, erosions and mucosal ulceration. Pharmacologic or immunologic inhibition of neutrophil function and mediator release attenuates the injury and dysfunction associated with experimental or human IBD (Harris et al

1992, Grisham 1993). Because neutrophils can be observed not only in the gut interstitium but also in the crypt lumen (e.g. crypt abscesses) of the inflamed bowel, it would appear that migration of these cells across the endothelial as well as the epithelial cell barrier is required, and may be important in the development of gut injury and dysfunction.

**Infective agents – bacterial and viral:**

Major advances in molecular biology have produced innovative techniques for detecting microorganisms present in very low concentrations. This has resulted in the identification of many new bacteria and viruses. As a result, many diseases that were formerly thought to be idiopathic have now been proved to be caused by infectious agents. An infectious etiology for inflammatory bowel disease has long been postulated, partly prompted by the similarity of the features of inflammatory bowel disease with classic infectious enteritis. Crohn et al, in their original description of regional ileitis, later called Crohn's disease, were unable to identify a causative agent using cultures and staining techniques (Crohn et al 1932). A highly significant concentration of cases of Crohn's disease were found in Cardiff, South Wales (Mayberry and Hitchens 1978). A cluster of Crohn's disease was present in a small Cotswolds village (Allan et al (1986). Four Crohn's disease cases were developed in adult women who had lived in close contact

during their teenage years, and suggested that the illness was caused by an infectious agent with a long latent period (Reilly and Robinson 1986). Crohn's disease and ulcerative colitis have been shown to develop in married couples, suggesting an infectious etiology (Lobo et al 1988, Batty et al 1994). Crohn's disease in a married couple and their four children, with three of the cases occurred within 10 months (Darchis et al 1989).

**Infective agents – mycobacteria:**

A link between mycobacterial infection and Crohn's disease was first postulated over 80 years ago, yet despite the application of increasingly sophisticated laboratory techniques, the issue remains unresolved. Chronic interstitial enteritis is linked with the pathology not only to tuberculous bowel disease, but also to an animal chronic enteritic infection termed Johne's disease, caused by *Mycobacterium paratuberculosis* (Dalziel 1913). Crohn himself noted the similarities to ileal tuberculosis when he described 14 cases of a granulomatous disease involving the small intestine (Crohn et al 1932). *M. paratuberculosis* has historically been suspected of being involved in Crohn's disease, largely because it has long been recognized to cause an infectious ileitis in ruminants, termed Johne's disease. *M. paratuberculosis* itself belongs to the *Mycobacterium avium* group of mycobacteria, but differs from *M. avium* in being mycobactin-dependent on

culture. *M. paratuberculosis* is the Johne's disease bacillus that has been found only in animals with Johne's disease or in humans with Crohn's disease.

#### **Mucosal defenses:**

MUC2 is highly expressed in the jejunum, duodenum, ileum and colon, and to a lesser extent in the gallbladder and bronchus (Ho et al 1993, Gambús et al 1993). MUC3 is expressed in the jejunum, ileum, colon and gallbladder (Gambús et al 1993). In both inflammatory and neoplastic colonic diseases disorders in mucin biosynthesis are observed which result either from differences in the relative expression of the different apomucins or from changes in the synthesis of the oligosaccharide side-chains, each of which may affect the physical properties of the viscous mucus gel.

Ulcerative colitis is associated with both qualitative and quantitative alterations in mucin glycoproteins.

#### **Absorption of fluids and electrolytes:**

In health, the colon conserves the electrolytes and water in chyme so effectively that desiccated stools can be stored and eliminated later, at a socially convenient time. Frequent, uncontrolled evacuation of blood, mucus and liquid feces by patients with inflammatory bowel disease (IBD) represents a dramatic change from this orderly function, and indicates a



major disturbance of colonic function. Study of the normal function of the colon in humans is complicated by regional differences in absorption, a complex and poorly understood pattern of motility, and the largely unexplored relationships between the host and the resident bacterial flora. The pathophysiology of IBD and its effects on colonic function are still obscure. The uncertain effects of mucosal inflammation cannot be ignored. Thus, in addition to deranged absorption, IBD might be anticipated to provoke exudation and possibly other mechanisms of fluid secretion. Inflammatory disease may therefore lead to diarrhea by several mechanisms. Blood, serum and mucus may exude from sites of ulceration or inflammation. When exudation is prominent, as in distal proctitis, rectal bleeding and diarrhea (with frequent, small, sometimes hard stools) may be a feature even though the fecal excretion of water is normal (Lennard-Jones et al 1962). Another mechanism of diarrhea is the accumulation of fluid in the lumen, as a result of active secretion by the diseased colon (Hawker et al 1980).

**Pathophysiology of gastrointestinal motor disturbances in IBD:**

The tonic and phasic contractions of the intestinal smooth muscle control the rate at which material travels along the gut before it is finally expelled through the anus. In theory, excessive propulsive motor activity would

accelerate the transit of material, producing diarrhea, whereas a preponderance of mixing or segmental contractions could result in delayed transit and constipation. The mechanisms responsible for the bowel disturbance in inflammatory bowel disease are presumably caused by interactions of abnormal motor activity (Read & Johnson 1983) and epithelial transport (Steadman & Philips 1990). Impaired colonic absorption of sodium and chloride (Duthie et al 1964, Harris & Shields 1970, Rask-Madsen et al 1973, Hawker et al 1980), and excessive secretion of fluid and electrolytes (Archampong et al 1972, Edmonds & Pilcher 1973) are thought to be responsible for the liquid diarrhea observed in patients with active colitis. Failure of the diseased colon to salvage unabsorbable carbohydrate may also contribute to the diarrhea in some patients (Montgomery et al 1968, Read 1982, Rao et al 1987). Until recently, disturbances of intestinal motor activity in ulcerative colitis and Crohn's disease were less well understood.

**Mucosal metabolism and proliferation:**

The efficient and effective function of intestinal epithelium as a barrier between luminal macromolecules and microorganisms on the one hand, and the internal milieu on the other, is central to the health of the intestines and of the organism as a whole. Failure to achieve an effective barrier has

several deleterious consequences. For example, the lamina propria may be exposed to luminal macromolecules, many of which are potent inducers of immunoinflammatory events; the epithelium may have a reduced capacity to perform its absorptive/secretory roles; and tissue fluid and cells may cross the epithelium and be lost to the lumen. An understanding of the biology, pathobiology and modulation of injury to, and maintenance and repair of, the epithelial barrier may offer insight into pathogenic mechanisms and potential therapeutic strategies in inflammatory bowel disease (IBD).

## **II-4: COLONIC POLYPS AND MALIGNANT NEOPLASMS:**

### **Colonic Polyps:**

Colonic polyps may be divided into two major groups: neoplastic (the adenomas and carcinomas) and non-neoplastic. The adenomas and carcinomas share a common characteristic-cellular dysplasia-but they may be subdivided according to the relative contribution of certain microscopic features. The non-neoplastic polyps may be grouped into several distinct categories, including hyperplastic polyps, and others.

### **Neoplastic Polyps (Adenomatous and Malignant Polyps):**

Adenomatous polyps are tumors of benign neoplastic epithelium that may be either pedunculated, attached by a narrow stalk, or sessile, attached by a broad base with little or no stalk (Itzkowitz 1996, Lev 1990). The neoplastic nature of adenomas is apparent by histologic examination of their glandular architecture. By definition, all colorectal adenomas are dysplastic. Adenomatous epithelium, is characterized by abnormal cellular differentiation and renewal, resulting in hypercellularity of colonic crypts. Adenomas have classically been categorized into three size groups: less than 1 cm, and greater than 2 cm (Mutto et al 1975). Overall, most adenomas are smaller than 1 cm. The three principal features that correlate with

malignant potential for an adenomatous polyp are size, histologic type and degree of dysplasia.

**Prevalence of Adenomas:**

The prevalence of adenomatous polyps is affected by two major factors: the inherent risk for colon cancer in a population and the age of the subjects. Although the frequency of colonic adenomas varies widely among populations, it tends to be higher in populations at greater risk for colon cancer (Correa 1978). Age is perhaps the single most important independent determination of adenoma prevalence (Clark et al 1985, Edie & Stalsberg 1978, Stemmermann et al 1973, Vatn & Stalsberg 1982).

The distribution of adenomatous polyps within the colon differs, depending on the method of investigation. In autopsy series, which presumably represent the normal distribution in asymptomatic persons, adenomas are uniformly distributed throughout the colorectum.

In the older age groups, particularly in persons older than age 60, both autopsy and colonoscopic studies of symptomatic as well as of asymptomatic subjects (DiSario et al 1991, Granqvist 1981) demonstrate a proximal shift in adenoma distribution. This phenomenon may have importance for deciding appropriate colon cancer screening approaches.

### **Gastrointestinal Polyposis Syndromes:**

Gastrointestinal polyposis refers to the presence of multiple polypoid lesions throughout the gastrointestinal tract. These lesions may be characterized by their distribution in the gut or their tendency to be inherited within a family, but they are best understood in the context of histopathology. Many of the polyposis syndromes are distinctive entities clinically and pathologically that have been sorted into recognizable categories over the past century or so (Rustgi 1994).

### **Malignant Neoplasms of the Large Intestine:**

Cancer of the colon and rectum (colorectal cancer) is a major cause of cancer-associated morbidity and mortality in North America, Europe, and other regions where life-styles and dietary habits are similar. The fourth most common newly diagnosed internal cancer overall in the United States (behind cancers of the prostate, breast, and lung), colorectal cancer currently constitutes 9% of new cancer cases in men and 11% in women. Currently in the United States the prevalence of colorectal cancers in whites is higher in the caecum and ascending colon (22% in males, 27% in females) and in the sigmoid colon (25% in males, 23 in females) (Ziegler & De Vesa 1986).

### **Fat, Bile Acids, and Bacteria:**

Several lines of evidence suggest that diets containing large proportions of fat predispose to colorectal cancer, especially in the descending and sigmoid colon. Colon cancer rates are high in populations whose total fat intake is high and are lower in those who consume less fat. On average, fat (saturated plus unsaturated) comprises 40% to 45% of total calorie intake in Western countries with high rates of colorectal cancer, whereas in low-risk populations fat accounts for only 10% to 15% of dietary calories (Shike et al 1990).

### **Fiber**

Epidemiologic and animal studies suggest that dietary fiber protects against the development of colon cancer. Dietary fiber is plant material that resists digestion by the upper gastrointestinal tract. Increased fiber intake, in the form of whole wheat and rye bread, also reduces the concentration of fecal secondary bile acids and fecal mutagens in healthy subjects (Heilbrun et al 1989, Reddy et al 1987).

### **Familial colon cancer:**

It has become increasingly clear that genetic predisposition plays a role in a substantial number of colorectal cancers. Although it is convenient to categorize cancers into hereditary (or familial) and nonhereditary (or

sporadic) types, it is more appropriate to assume that all cancers have genetic components, which may be inherited or acquired, to varying degrees.

### **Predisposition to colorectal cancer:**

#### *Age:*

The risk of developing colorectal cancer rises sharply after age 40 in the general population, with 90% of cancers occurring in persons aged 50 years and older. In fact, a 50 year-old person has approximately a 5% chance of developing colorectal cancer by age 80 years and a 2.5% risk of dying from the disease (Atkin et al 1992).

#### *Adenoma*

Present evidence strongly indicates that the majority of colorectal cancers arise from pre-existing adenomas. The risk of colorectal cancer increases with the number of adenomas. Despite the potential for adenomas to evolve to carcinomas, the actual risk is unknown.

#### *Carcinoma*

People with one colorectal carcinoma have an increased risk of harboring a second carcinoma (synchronous carcinomas).

#### *Family History*

The risk of colorectal cancer in first-degree relatives of those with sporadic colorectal cancer is increased two-to-three fold. The risk is higher when



adenoma or carcinoma has occurred in a relative at an early age or when more than one relative has had carcinoma (Neugut et al 1995, Fuchs et al 1994, St. John et al 1993, Winamar et al 1996).

#### *Inflammatory Bowel Disease*

Patients with idiopathic inflammatory bowel disease (ulcerative colitis, Crohn's disease) are at increased risk for developing adenocarcinoma of the colon (Provengel et al 1995, Nugent et al 1991, Lennard-Jones et al 1990, Ekbon et al 1990, Katzka et al 1983).

#### **Tumor Pathology, Natural history and Staging:**

##### *Gross Features*

The gross morphologic features of adenocarcinoma in the large bowel depend on the tumor's site. Carcinomas of the proximal colon, particularly those of the caecum and ascending colon, tend to be large and bulky, often outgrowing their blood supply and undergoing necrosis. This polypoid configuration may also be found elsewhere in the colon and rectum. In the more distal colon and rectum, tumors more frequently involve a greater circumference of the bowel, producing an annular constriction or "napkin-ring" appearance.

### *Histology*

Carcinomas of the large bowel are predominantly adenocarcinomas, which form moderately to well-differentiated glands and secrete variable amounts of mucin. In approximately 15% of tumors, large lakes of mucin containing scattered collections of tumor cells are present. These mucinous or colloid carcinomas are most frequent in patients with hereditary non-polyposis related carcinomas, in the setting of ulcerative colitis, and in patients whose carcinomas occur in early age. Schirrhous carcinomas are uncommon and are characterized by sparse gland formation, with marked desmoplasia and fibrous tissue surrounding glandular structures. Cancers other than adenocarcinomas account for fewer than 5% of malignant tumors of the large bowel.

### **Natural History and Staging:**

Colorectal cancers begin as intramucosal epithelial lesions, usually arising in adenomatous polyp or glands. As cancers grow they become invasive, penetrating the muscularis mucosae of the bowel and invading regional lymph nodes, adjacent structures, and distant sites. Although adenocarcinoma of the colon and rectum grow at varying rates, they most often have long periods of silent growth before producing bowel symptoms. The mean doubling time of colon cancers determined radiographically in

one study was 620 days. Patterns of spread depend on the anatomy of the individual bowel segment as well as its lymph and blood supply.

The TNM classification for human carcinomas is based in part on the observation that, for most cancers, tumor size correlates with local and distant spread and, thus, with prognosis. Patients with exophytic or polypoid tumors appear to have better prognosis than those with ulcerating or infiltrating tumors.

## **II-5: CLINICAL FEATURES:**

### **Ulcerative colitis:**

The leading initial symptom of ulcerative colitis is diarrhea with blood and mucus, sometimes with pain. Fever and weight loss are less frequent. Extraintestinal symptoms can be an initial manifestation or can occur later in the course of the disease. In proctitis, rarely constipation can be the initial symptom. Eighty percent of the patients have only proctitis or proctosigmoiditis, and only 20% have extensive colitis. However, in about 50% of patients with initial proctosigmoiditis, proximal extension occurs later, and in some patients the opposite takes place. 50 to 70% have a relapse during the first year after diagnosis. The relapse rate is higher in the younger patients and seems to decrease with increasing age.

### **Crohn's disease:**

The clinical presentation of Crohn's disease is determined largely by the site of intestinal inflammation. The cardinal symptoms are of diarrhea (70-90%), abdominal pain (45-65%), and weight loss (65-75%), with frequencies reflecting the predilection of Crohn's disease for the ileocecal region. Extraintestinal manifestations occur in up to one-third of the patients.

Examination findings may include a tender fullness in the right ileac fossa and low-grade fever.

Patients presenting with a severe flare-up look ill, may be febrile (temperature  $> 38^{\circ}$  C) and tachycardia, and have laboratory evidence of inflammation or infection (elevated erythrocyte sedimentation rate, C-reactive protein level, and white cell count): they should be admitted to hospital for treatment. However, the picture often is much less dramatic, and Crohn's disease can be nonspecific in its presentation, with little more than malaise and weight loss, for example. Such cases can present a major diagnostic challenge.

The time course of the illness is particularly important, as many patients will have experienced remitting and relapsing symptoms over a number of years before seeking medical attention, or may have been misdiagnosed, most commonly with irritable bowel syndrome.

The presence of a positive family history of inflammatory bowel disease markedly increases the prior probability of a diagnosis of Crohn's disease: 10 or 20% of Crohn's disease patients have an affected first-or second-degree relative, compared to less than 1% of the general population.

### **Diverticular disease:**

The clinical presentation of diverticular disease varies depending on the pathologic process, and many patients present electively with lower abdominal pain, distention, and altered bowel habit. Clinical examination is usually unrewarding apart from findings of nonspecific tenderness and occasionally palpable colon in the left iliac fossa. The underlying diverticular disease is demonstrated by endoscopy and radiology to exclude malignant disease.

### **Polyps of the large intestine:**

Most polyps are asymptomatic, but they may cause macroscopic as well as occult bleeding. Bleeding occurs with increasing frequency when the polyp is over 1 cm in diameter. Juvenile polyps have a rich blood supply and may be symptomatic more often than adenomatous polyps; red bleeding being most frequent, because of ulceration, but also because of torsion of the stalk with sloughing of the polyp. A child may present with a red protrusion from the anus, whereas invagination because of the polyp is rare.

More symptomatic adenomatous polyps are in the left colon and rectum, and the bleeding therefore is red, mixed with feces, or on the surface. Right-side adenomatous polyps 1 cm or more in diameter may result in a positive guaiac test. Bowel habits may change when the polyp becomes large ( $\geq 3$

cm); rarely, invagination caused by lipomas, adenomatous polyps, or juvenile polyps may occur, causing peristaltic cramp. A dynamic ileus may be seen in patients with very large polyps in the left colon.

**Colorectal cancer:**

In approximately one-fifth of all cases of colorectal cancer the patient has sought emergency care because of constipation, colonic obstruction, peritonitis due to perforation of the abdominal cavity, or excessive bleeding. These patients must be treated immediately, often with an emergency laparotomy, which provides correct diagnosis at surgery.

A small group of patients admitted to the hospital due to anorexia or extreme fatigue are found on subsequent investigation to have metastatic colorectal cancer.

The majority of patients with colorectal cancer, however, present with vague symptoms, like a change in bowel habits, diffuse abdominal pain, or discomfort. All patients should also be told to seek medical attention as soon as they notice blood in their stool, whether or not they believe the blood to be related to hemorrhoids. The golden rule is that all patients, regardless of age, with anorectal discomfort or symptoms in this area should undergo digital examination of the anal canal and rectum and a rigid sigmoidoscopy.

## II-6: IMAGING STUDIES:

### **Crohn's Disease:**

The choice between endoscopic and radiologic modalities for imaging the bowel in Crohn's disease may not be clear-cut in many cases. Each has distinct advantages and disadvantages, and the decision as to which to use must be dictated by each patient's circumstances.

A plain abdominal radiograph can provide much useful information in the assessment of an acutely unwell patient with Crohn's disease. Small bowel obstruction may be evident as dilated loops of bowel, possibly with fluid levels; displacement of bowel loops may suggest an inflammatory mass in the right iliac fossa; and colonic inflammation causing mucosal edema may appear as thickening of the bowel wall.

A double-contrast barium enema shows the extent and activity of colonic disease and may be preferable to colonoscopy as a means of demarcating fistulae and for visualizing the mucosa proximal to strictures. The features of Crohn's disease are essentially as described for the small bowel enema, though differentiating inflamed or scarred mucosa from malignant infiltration may be difficult. The <sup>111</sup>Indium-labeled white cell scan may identify the site of an intra-abdominal abscess; in some centers it is used in



preference to barium radiology or colonoscopy to detect and delineate the extent of intestinal inflammation.

Endoscopic assessment of the intestinal mucosa together with targeted biopsy of macroscopically abnormal regions is a valuable tool for the gastroenterologist in diagnosing and assessing Crohn's disease activity.

**Ulcerative colitis:**

Depending on the stage of the disease, endoscopy reveals reddening of the mucosa, increased vulnerability, mucosal bleeding, irregular ulcers, pseudopolyps, granularity, and loss of vascular architecture. The changes are continuous, and the rectum is always involved if no effective local treatment has been applied. Abdominal transcutaneous ultrasonography may demonstrate bowel wall thickening of the involved area, but it is nonspecific. The same applies for computed tomography and magnetic resonance imaging.

Since the advent of endoscopy, double-contrast barium enema is rarely used. It does, however, show typical findings and can be applied if endoscopy is not possible or is not accepted by the patient.

**Diverticular disease:**

In the elective situation rigid or flexible sigmoidoscopy should precede visualization of the colon by double-contrast barium enema. Alternatively,

colonoscopy may be preferred to provide more direct assessment of the mucosa, although in diverticular disease severe spasm, fibrosis, and tortuosity of the sigmoid loop make passage of the colonoscope more difficult. For emergency admissions plain radiography of the abdomen and chest often provide indirect evidence of major inflammation: pneumoperitoneum, bowel distention, and abnormal soft tissue shadows. In acute diverticulitis bowel thickening, mucosal edema, irregularity, and occasional extravasation of contrast may be observed. Leakage of contrast, if present, is usually localized within a communicating abscess cavity, but free perforation into the peritoneal cavity is occasionally seen. Ultrasound scanning can be useful in acute diverticulitis and is routinely used in some centers. However, operator variability reduces its general applicability as the first investigation, and definitive assessment of the bowel with barium enema or colonoscopy is also required.

**Polyps of the large intestine:**

Most polyps are detected by endoscopic and radiologic investigations during examinations made because of symptoms suggesting inflammatory or neoplastic disease, but not caused by the polyps detected. The diagnostic accuracy of an ordinary barium enema is low; the double-contrast barium enema (DCBE), however, is nearly as accurate as flexible endoscopy when

the polyps are  $\geq 1$  cm in diameter. Diverticular may be mistaken for polyps and vice versa. Bowel preparation is necessary before all examinations of the large bowel. Conventional enemas may be used, or a large amount of fluid with polyethylene glycol can be ingested perorally. The most extensive preparation is needed for complete colonoscopy. In a well-prepared bowel the flexible sigmoidoscope may pass to the left flexure in 25% of the patients, provided that no anatomic obstacles are present, and the sigmoid colon may be visualized in 80% of the patients.

#### **Colorectal Cancer:**

Barium enema, preferably double-contrast barium enema, has for years been the visualization tool of choice in patients with symptoms suggesting colorectal cancer. It should be emphasized, however, that it is extremely difficult to visualize low rectal cancers on a radiograph.

Another choice is to do a colonoscopy as the first examination. As with colonoscopy used in screening situation, this examination permits therapeutic maneuvers, like polypectomies and biopsies. However, colonoscopy may be more painful for the patients than a barium enema, and even trained person may find it impossible to reach the caecum. Since there is no real difference to the diagnosis potential of these two imaging techniques when they are used properly, the main question is how to decide

## II-7: ULTRASONOGRAPHY OF COLON

### **Basic Principles:**

Gastrointestinal tract sonography is frequently frustrating and always challenging. Gas content within the gut lumen can make visibility difficult and even impossible, intraluminal fluid may mimic cystic masses, and fecal material may create a variety of artifacts and pseudotumors. Nevertheless, normal gut has a reproducible pattern or “gut signature”, and a variety of gut pathologies create recognizable sonographic abnormalities. In addition, in a few conditions-such as acute appendicitis and acute diverticulitis-sonography may play a major primary investigative role. Further, endosonography, performed with high-frequency transducers in the lumen of the gut, is an increasingly popular technique for assessing the esophagus, stomach, and rectum.

### **Transabdominal sonography of colon:**

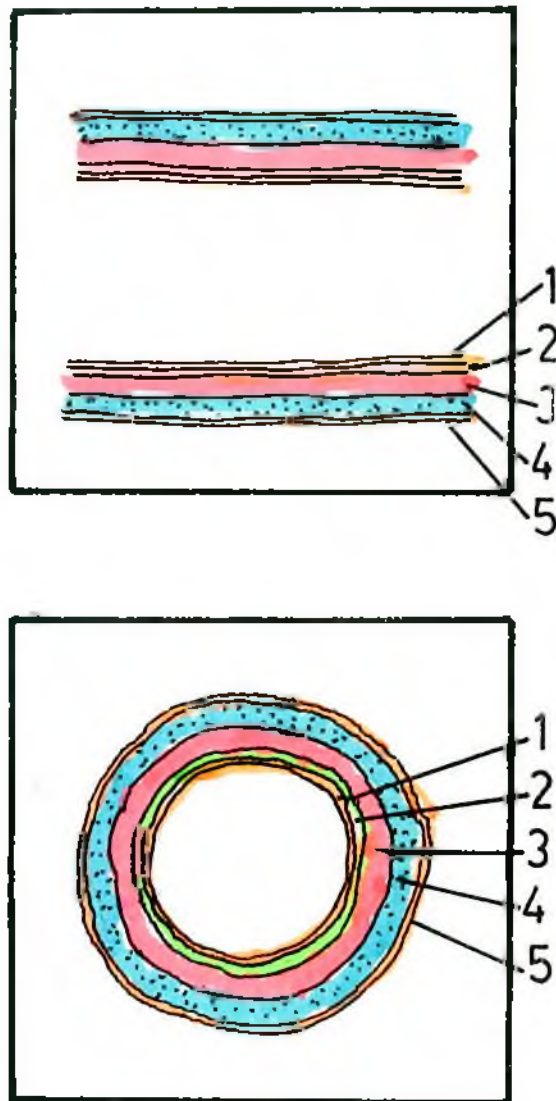
The gut is a continuous hollow tube with four concentric layers. From the lumen outward, they are: (1) mucosa, which consists of an epithelial lining, loose connective tissue or lamina propria, and muscularis mucosa; (2) submucosa; (3) muscularis propria, with inner circular and outer longitudinal fibers; and (4) serosa and adventitia.

These layers create a characteristic appearance or gut signature on sonography, where up to five layers may be visualized. The correlation of the histologic layer with the sonographic appearance are well established (Heyder et al 1987, Bolondi et al 1986, Kimmey et al 1989). The sonographic layers are alternatively echogenic and hypoechoic; the first, third, and fifth layers are echogenic; and the second and fourth layers are hypoechoic (Fig – II-6). On routine sonograms, the gut signature may vary from a bull's eye in cross-section, with an echogenic central area and a hypoechoic rim, to full depiction of the five sonographic layers. The normal gut wall is uniform and compliant, with an average thickness of 3 mm of distended and 5 mm if not.

#### *Gut Wall Pathology*

Gut wall pathology creates characteristic sonographic patterns. The most familiar sonographic appearance is the "target" pattern or 'pseudokidney' pattern (Lutz & Petzoldt 1976, Bluth et al 1979). When these patterns are identified, a pathologically significant lesion was found in more than 90% of patients. In both descriptions, the hypoechoic external rim corresponds to thickened gut wall, whereas the echogenic center relates to residual gut lumen or mucosal ulceration. The target and pseudokidney are the abnormal equivalents of the gut signature created by normal gut.

Figure No. – II-6



Longitudinal and transverse section of colon representing 5 layers seen on a sonographic image. 1 – Hyperechoic, 2 – Hypoechoic, 3 – Hyperechoic, 4 – Hypoechoic, 5 – Hyperechoic.

Gut wall masses, as distinct from thickened gut wall, may be intraluminal, mural, or exophytic, all with or without ulceration. They may be difficult to assign to a gastrointestinal tract origin if typical gut signatures, targets, or pseudokidneys are not seen on sonography.

Routine sonograms are best performed when the patient has fasted. A real-time survey of the entire abdomen is performed with a 3.5-and/or a 5MHz transducer in which any obvious masses or gut signatures are observed. The pelvis is scanned before and after the bladder is emptied. Areas of interest then receive detailed analysis, including compression sonography. Although this technique was initially described using high-frequency linear probes, 5-MHz convex linear and some sector probes work extremely well. The critical factor is a transducer with a short focal zone allowing optimal resolution of structures close to the skin. Slow graded pressure is applied. Normal gut will be compressed, and gas pockets displaced away from the region of interest.

*Inflammatory bowel disease:*

Inflammatory bowel disease includes Crohn's disease and ulcerative colitis. Although barium study and endoscopy remain the major tools to evaluate mucosal and luminal abnormality, sonography, like CT, may offer valuable additional information about the gut wall, the lymphnodes, the mesentery,

and the regional soft tissues (Seitz & Rettenmaier 1988). The chronic nature of inflammatory bowel disease, characterized by multiple remissions and exacerbations, is well assessed by a noninvasive, sensitive modality such as sonography. The degree of thickening of the gut wall and the frequent association of extraluminal disease make Crohn's disease the most optimally studied disorder. Baseline examination with follow-up predicts complications such as abscess, fistula, or obstruction; detects postoperative recurrence; and identifies patients who require more invasive imaging techniques (Sarrazin & Wilson 1996).

Sonography may be used in patients with Crohn's disease to document the classic features including gut wall thickening, strictures, creeping fat, hyperemia, mesenteric lymphadenopathy, and uncommonly mucosal abnormalities or complications including inflammatory masses (phlegmon or abscess), fistula, obstruction, perforation or appendicitis (Sarrazin & Wilson 1996).

Detect thickened loops of gut. This may be appropriate for initial detection, for detection of recurrence for determining the extent of disease, and in follow-up in the assessment of improvement (DiCandio et al 1986). Gut wall thickening is most frequently concentric and may be quite marked (Worlicek



et al 1987, Dubbins 1984). Wall echogenicity varies depending on the degree of inflammatory infiltration and fibrosis.

### **Endoluminal ultrasonography of colon:**

Since the introduction of endoluminal ultrasound for the evaluation of the lower gastrointestinal (GI) tract, it has assumed a greater role in the assessment of both inflammatory and neoplastic disease of the colon, rectum and anal canal. The ability to configure transducers with frequencies ranging from 7.0 to 20 MHz as either radial, linear or sector probes and incorporate them into endoscopes has enabled the sonologists to image multiple histologic layers within the wall of the lower GI tract (Rosch & Classon 1992, Alexander et al 1994)

#### *Instrumentation:*

The colon is usually imaged ultrasonographically during colonoscopy with a 7.5-12 MHz probe mounted on the end of an endosonoscope. Mechanical transducers produce ultrasound images by rotating elements in a radial fashion, generating a 90-360° image. Electronic transducers are motionless, relying on electrical impulses to create ultrasound images in a pie-shaped sector or rectangular (linear) configuration. Most of these transducers image in the transverse (axial) plane, except for the linear transducer, which produces a longitudinal image. With these transducer frequencies, sufficient

depth of penetration and resolution should be available to image the multilayered rectal wall and any adjacent structures such as blood vessels, lymph nodes and visceral organs.

Higher-frequency transducers (12.5-20 MHz) are now available that acquire exquisitely detailed images of the colorectal wall layers over a short distance of 2-3 cm.

*Preparation and techniques:*

For any endosonographic study of the lower GI tract, the transducer-containing portion of the probe is covered by a condom, which provides protection against fecal contamination. It also serves as a reservoir for water, which upon distention provides a pathway for the conduction of sound waves from the transducer to the target. Additional important benefits obtained from the water-filled condom include a means to artificially adjust the focal depth of the transducer by altering the water volume, distend the bowel lumen, and displace gas and residual fecal debris away from the area of interest, reducing the potential for confusing pseudolesions. Distention of the condom is best achieved by injecting various amounts of degassed water from a catheter-tipped syringe through an extension tube attached to an injection port on the probe. Most patients are examined in the left lateral

decubitus position with their knees toward their chest and their buttocks over the table edge.

*Endosonographic anatomy:*

Most investigators now agree that at least five layers of the wall can be detected with endosonography, depending on the level at which the colon or rectum is imaged and the frequency utilized (Wang et al 1987, Orrom et al 1990, Hildebrandt et al 1986, Alexander 1994). Sonographically, each layer is represented by either a thin hypoechoic (dark or echo-poor) or hyperechoic (bright or echo-rich) band. The initial layer imaged is the echogenic wall of the water-filled condom as it interferes with the surface of the colonic mucosa. The next hypoechoic band represents the mucosa in both the colon and rectum, although some investigators believe that it may be a composite of both the mucosa and muscularis mucosa. The third sonographic layer is the hyperechoic submucosa which is the thickest layer in the wall. The fourth sonographic layer is the hypoechoic smooth muscle of the muscularis propria. The fifth and final layer is the hyperechoic colonic serosa.

*Malignant colorectal neoplasms:*

The typical colorectal cancer imaged with endosonography will appear as a hypoechoic mass, although, on occasion, it may have a mixed echotexture

with both bright and low-level internal echoes. More recently, investigators utilizing endorectal Doppler probes have detected color flow and pulsed Doppler signals centrally and peripherally within the blood vessels feeding tumor masses (Alexander et al 1994).

*Benign mucosal and submucosal tumors:*

The small benign adenoma and polyp may not be distinguished from the early mucosal carcinoma. Likewise, the large villous adenoma may contain clinically unrecognized cancer, which is not detected by random superficial biopsies. The key point is recognizing that the lesion is not invading the hyperechoic submucosa, which is imaged as a continuous unbroken line (Deen et al 1995, Hulsmans et al 1992).

The most frequently encountered submucosal tumor is the leiomyoma, which arises from the hypoechoic muscularis, and is usually described as a smooth-margined, hypoechoic mass (Detry et al 1993, Hulsmans et al 1993). Size may be the only feature that distinguishes this tumor from the larger leiomyoblastoma and leiomyosarcoma.

*Inflammatory bowel diseases:*

Ulcerative colitis has been classified using endosonography, into five different types (Shimizu et al 1992). Classification criteria were based on thickening of the superficial versus deep layers and distensibility. The role

that endosonography plays in the diagnosis and management of the patient with ulcerative colitis remains unclear.

*Diverticula:*

Diverticula are mucosal outpouches through a weakened muscularis propria. They may become obstructed and inflamed, leading to perforation or abscess formation. In asymptomatic patients, diverticula are rarely noticeable on the sonogram. However, when inflamed, diverticula are seen as outpouchings protruding beyond the lumen of the gut into or beyond the thickened colonic wall.

*Future potential:*

Three-dimensional ultrasonography has been utilized in the study of the heart and, recently, this technology has been incorporated into endosonographic probes to evaluate the prostate (Rankin et al 1993, Litrup et al 1987). Although this technology has been applied to the upper GI tract, it could have applications in the evaluation and staging of colorectal lesions. A method have been developed to use simultaneous high-frequency ultrasound and real time manometry to evaluate the physiologic processes involved in the peristaltic contraction of the esophagus (Miller et al 1995). This same methodology could be used in the anal canal to better define

normal anal physiology and to evaluate various disease processes in the anal canal.

*Conclusion:*

Endosonographic technology has progressively improved over time and now provides diagnostic information not available from the clinical examination alone. Compared to CT and MRI, it is less costly and, unlike contrast radiography and CT, it does not utilize ionizing radiation.

In conclusion, progressive improvements of ultrasound technology, especially endoluminal ultrasound, have greatly aided diagnostic evaluation of the lower GI tract. As an easily performed, relatively inexpensive noninvasive technique, endosonography has improved the assessment of the colorectum and anal canal and has shown promise in providing more useful information to the clinician in the diagnosis and management of malignant and nonmalignant diseases in the lower GI tract.

---

**MATERIALS**  
**CHAPTER 3**

### **III: MATERIALS**

#### **III-1: PERIOD AND PLACE OF STUDY:**

During a 23 month's period from July 1999 to May 2001 patients were referred to the Institute of Nuclear Medicine, bangabandhu Sheikh Mujib Medical University, Shahbagh, Dhaka and the Training & Research Centre, the Bangladesh Institute of Ultrasound in Medicine & Research of the University of Science & Technology Chittagong, Eastern Plaza, Dhaka. The patients were referred from 18 departments of 7 Institutions in the Dhaka area: Dhaka Medical College Hospital, Mitford Hospital, Institute of Post Graduate Medicine & Research and Bangabandhu Sheikh Mujib Medical University, BIRDEM Hospital, Holy Family Red Crescent Hospital, Bangladesh Medical College and 200 Beded General Hospital of Narayanganj. Most of the patients were referred from surgical and medical outpatient departments and inpatient departments. More over about 20 Family Physicians referred patients from their private practicing chambers.



### **III-2: NUMBER OF PATIENTS:**

Materials comprised of 456 patients with either clinically suspected colon diseases or normal control. 67 patients had two examinations giving a total 523 examinations. The clinical situation prior to the second ultrasound examination in these 67 patients was a new one in each case. For practical reasons, the patient was therefore considered as a new patient at the time of the second examination. 15 patients were dropped from the study due to inadequate preparation and the patients refused take further preparations. Finally 508 patients were included into the study.

Out of the 508 patients, 457 [89.96%] were male and 51 [10.04%] were female. Age ranged from 14 to 78 years, mean being 48.73 years. The age and sex distributions were presented in the Chart – III-1. All the patients were well informed about the procedure of hydrocolonic sonography. Written consent was obtained from each of the patients in front of a witness. A history sheet was filled up by the information obtained from each of the patients. The study protocol was approved by the Faculty of Postgraduate Medical Science & Research of Dhaka University. All the 508 patients included into the study were subdivided into Group-I and Group-II.

**Group-I:**

Total 368 patients were included into Group-I. Out of 368 patients, 328 [89.13%] were male and 40 [10.87%] were female. Age ranged from 14 years to 75 years, mean being 47.88 years. The age and sex distributions were presented in the Chart – III-2. All the patients were examined by double contrast barium x-ray with the same preparations in the same day. If the examination was deferred to a next day, a separate preparation was given.

**Group-II:**

Total 140 patients were included into Group-II. Out of 140 patients, 129 [92.14%] were male and 11 [7.86%] were female. Age ranged from 15 years to 78 years, mean being 53.79 years. The age and sex distributions were presented in the Chart – III-3. All the patients were examined by double contrast barium x-ray with the same preparations in the same day. If the examination was deferred to a next day, a separate preparation was given. All the patients were examined either by colonoscopy and biopsy and or surgery. All the diagnosis obtained by hydrocolonic sonography and double contrast barium enema x-ray were reconfirmed by colonoscopy and or surgery.

**Criteria of inclusion and exclusion:**

Patients were selected randomly either with complains of large intestine as follows:

- a. Diarrhea
- b. Pain abdomen
- c. Blood with stool
- d. Mass in the abdomen
- e. Constipation

Or the patients had no complain related to the colon diseases. Patients were examined by hydrocolonic sonography to detect normal pattern of the colon and pathological changes of the colon.

Patients were excluded from the study due to;

- a. Acute illness
- b. Acute diarrhea
- c. Suffering from acute infectious diseases
- d. In mild to moderate illness, the examination was discontinued for the time being and re-examined in a second session.
- e. No absolute contraindication was noticed.

Chart – III-1

**Total patients = 508**

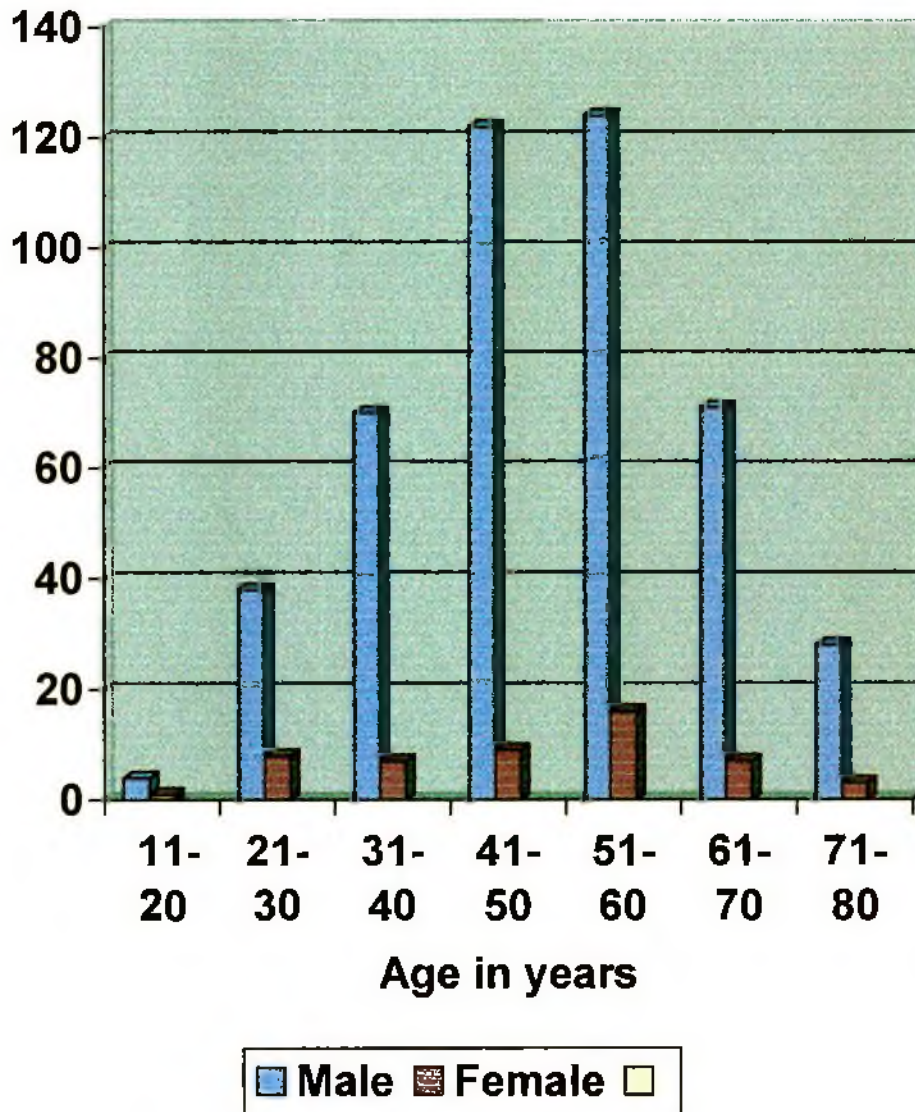


Chart – III-1: Showing age and sex distributions of total 508 patients included in the study. Male 457 [89.96%], Female 51 [10.04%]

Chart – III-2

**Total patients = 368**

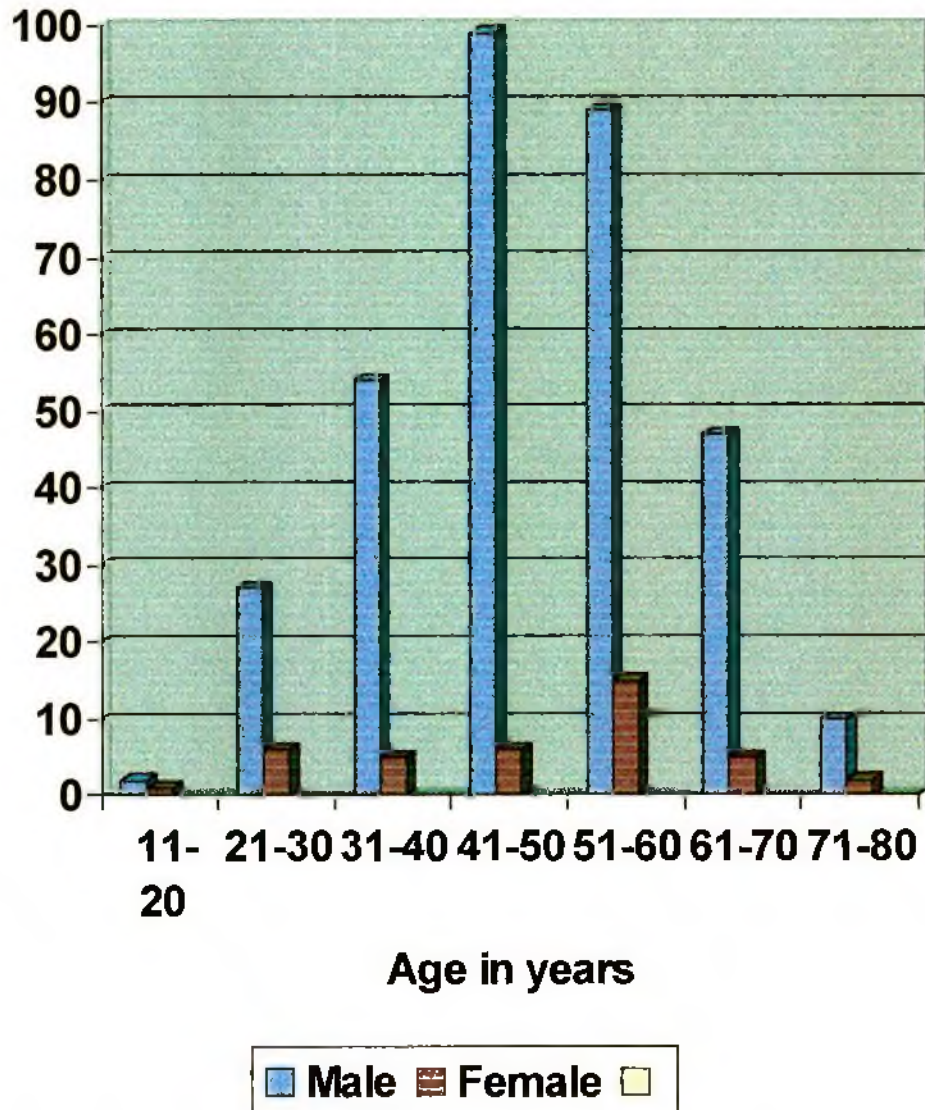


Chart – III-2: Showing age and sex distributions of 368 patients included in the Group - I. Male 328 [89.13%], Female 40 [10.87%].

Chart – III-3

**Total patients = 140**

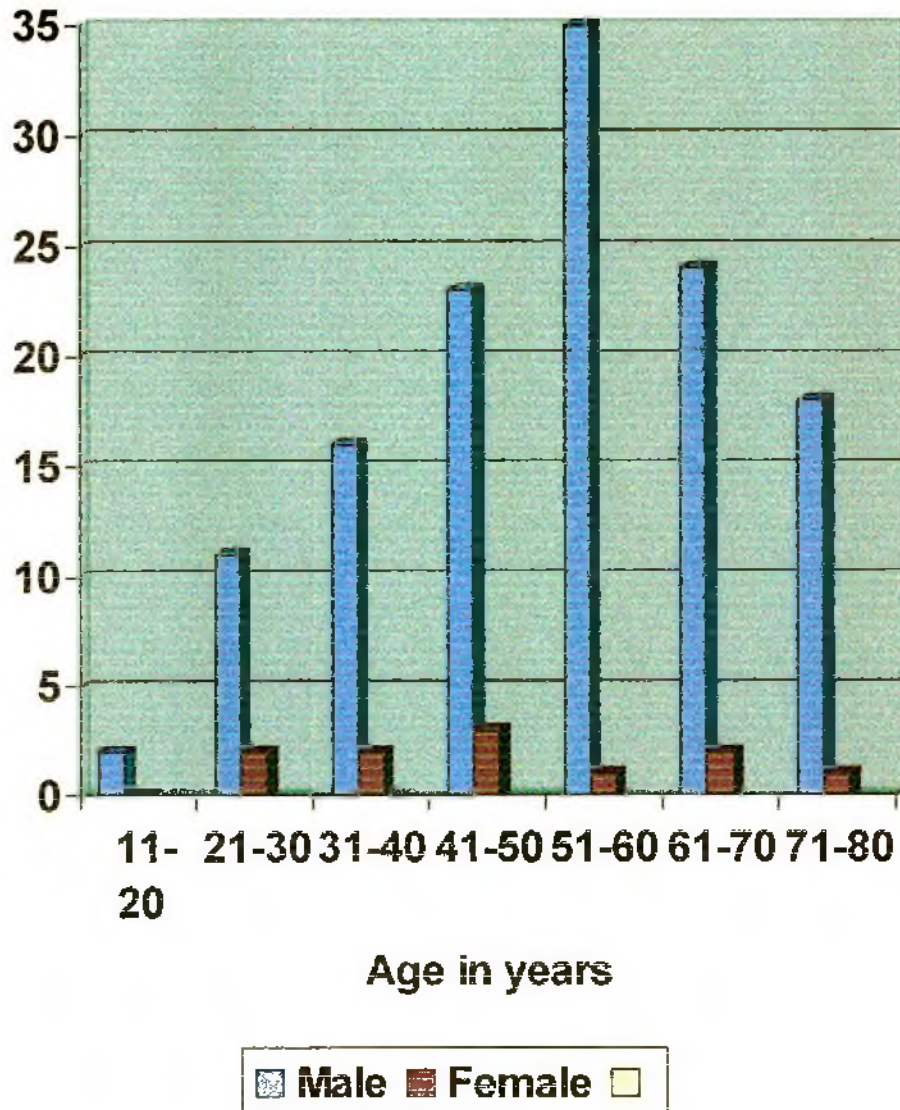


Chart – III-3: Showing age and sex distributions of 140 patients included in the Group - II. Male 129 [92.14%], Female 11 [7.86%].

### III-3: WATER AS CONTRAST AGENTS:

In hydrocolonic sonography normal tap water is used as contrast agent. When ultrasound beam passes through any medium it is reduced in intensity. This reduction in intensity is called attenuation. The degree of attenuation depends on the medium involved. An important parameter called the ultrasound attenuation coefficient is used to differentiate between attenuation properties of various media. Here is a table describing ultrasound coefficient of different biological medium.

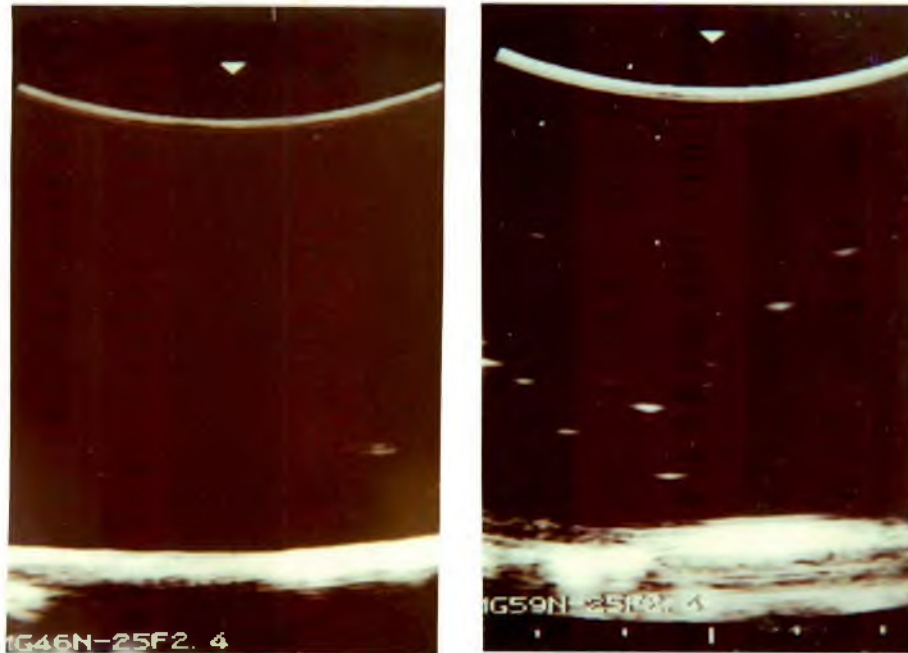
Material	Attenuation coefficient [dB/cm]
Lung	41
Bone	20
Air	12
Soft tissue average	1.0
Kidney	1.0
Liver	0.94
Brain	0.85
Fat	0.63
Blood	0.18
Water	0.0022

Water and blood have very low attenuation coefficients because of their low viscosity. These materials transmit ultrasound readily and are often called ultrasound windows.

Fresh tap water contains numerous small air bubbles in it. This small gas bubbles act as strong reflectors. To avoid this, tap water were kept two days in a container to make it bubble free [Fig – II-1].



Figure – III-1



Ultrasound Contrast effect of water: Ultrasonography of water kept two days in a container to make it bubble free was done. Complete anechoic images confirms thorough transmission ultrasound waves without any significant attenuation. The second image was taken from fresh tap water. Small echogenic spots indicated presence of small gas bubble, not suitable for hydrocolonic sonography

### **III-4: ULTRASOUND INSTRUMENTS**

#### **General considerations:**

Ultrasound was introduced into diagnostic imaging in the early 1960s. However it took approximately 20 years for it to develop into a principal diagnostic imaging modality. Its inherent advantages – relatively low cost, non-invasive and easily conducted examinations – have led to an ever increasing demand for ultrasound technology. Today diagnostic ultrasound stands as the first line of diagnostic imaging for many patients.

Ultrasound imaging technology started its journey as static B-scanner. The principle drawback was the time required to construct an image. An improvement over B-scanning is real-time imaging. The term real-time is borrowed from computer science and implies instant computation or, as in diagnostic ultrasound, instant imaging. Real time imaging becomes the imaging mode of choice with the introduction of the digital scan converter and the microprocessors. The principal advantage of the real-time is the ability to obtain images instantly and continuously of both static and moving internal structures.

These devices have allowed almost instantaneous reception, processing, and display of pulse-echo information. Semiconductor memory has become so large and inexpensive that multiple microprocessors are now used allowing

wide control of focusing, steering, and amplification of the ultrasound beam. This computer technology also allows for increased information density, providing images of superior quality.

The ability to observe internal motion is important in hydrocolonic sonography. Peristalsis of colon can be demonstrated easily by real-time scanner. Polyp can be differentiated from wall adherent stool.

Water is instilled into colon through anus. Continuous abdominal sonographic examination of the colon was carried out with real-time imaging devices.

#### **Ultrasound instruments:**

*A) Aloka SSD-500:* Manufacturer: Aloka Co. Ltd. 22-1, Mure 6-chome, Mitaka-shi, Tokyo 181, Japan [Fig – III-2]. Probes: Electronic convex probe, Type No. UST-974-5, Main specifications 5MHz, 20R, 54 degree, Abdominal application from surface of body. Electronic convex probe, Type No. UST-934-3.5, Main specifications 3.5MHz, 60R, 60 degree, Abdominal application from surface of body.

*B) Sigma 21:* Manufacturer: Kontron Instruments S. A., Montigny Le Bretonneux, France. Probes: 3.5MHz Sector, Abdominal, Long focus family. 7.5MHz Sector, Transrectal, Bi-plane [Fig – III-3].

C] Aloka SSD-1100: Manufacturer: Aloka Co. Ltd. 22-1, Mure 6-chome, Mitaka-shi, Tokyo 181, Japan. Probes: Electronic convex probe, Type No. UST-974-5, Main specifications 5MHz, 20R, 54 degree, Abdominal application from surface of body. Electronic convex probe, Type No. UST-934-3.5, Main specifications 3.5MHz, 60R, 60 degree, Abdominal application from surface of body. Electronic linear probe, Type No. UST-5711-7.5, Main specifications 7.5MHz, 51mm, from the surface of the body [Fig – III-4].

Figure – III-2



Aloka SSD-500 ultrasound scanner and 5.0MHz curvilinear transducer mainly used in this study

Figure – III-3



Kontrone Sigma-21 ultrasound scanner with 3.5MHz sector scanner and 7.5MHz bi-planer, sector, transrectal transducers.

### **III-5: ULTRASOUND IMAGE RECORDING DEVICES**

#### **Video graphics printer:**

Video graphics printer provides a black-and-white hardcopy recording of an image displayed on monitor. Fuji video graphics printer is used to get the hardcopies of images [Fig – III-5]. Heat-sensitive paper acts as the receptor medium for video printers. The paper is supplied in long rolls of several hundred feet and is cut to size within the device. A video signal sent to the video printer is captured as one or two fields and stored in computer memory. As paper moves past the CRT, it is exposed line by line. The exposed paper is heat developed by passage over a mechanical heater. Contrast and brightness on the CRT and development temperature were adjusted optimally.

Quality assurance of the entire ultrasound, including the recording devices, was monitored periodically to ensure that the recorded image matched that of the monitor.

#### **Copying of the images:**

Usually a single copy of each of the desired images was done. Multiple copies for the thesis were done with the help of Nikon F3 camera. Photograph was taken by the author, but development of film and printing on glossy paper were done by commercial firm.

Figure – III-5



Fuji video graphic ultrasound recording device



### **III-6: ACCESSORY DEVICES**

#### **Enema can:**

This is an ordinary enema can used in Radiology department and procured from commercial firm [Fig – III-6]. It consists of a can, rubber tube and a hard catheter. Warm soap water was prepared in the centre using water heater.

#### **Instilling can:**

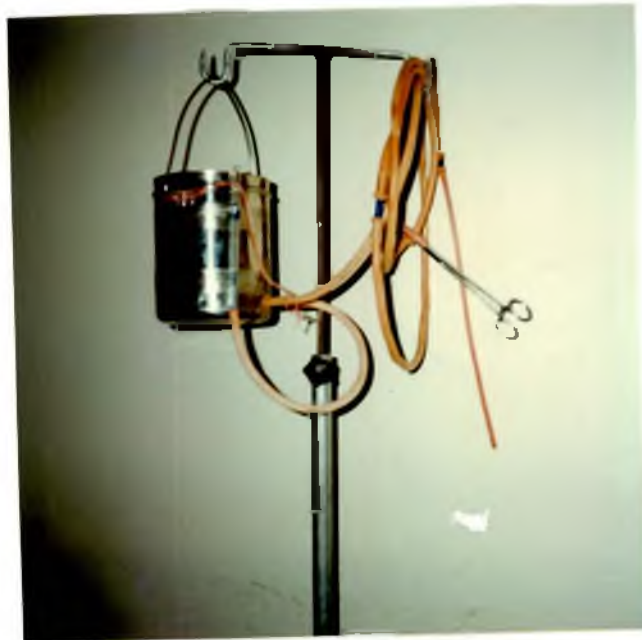
This is a special big sized can prepared for this purpose. In addition to delivery tube an extra glass tube is fitted at the side of the can so that level of water can be seen from outside. Rubber tubing is attached to the can, which ended with a catheter for introducing into anus. A forceps with catch is used to stop the flow. The can is hanged from a stand. Length of the stand is adjustable according to the need [Fig – III-7].

Figure – III-6



Conventional enema can used in radiological departments are used for preparing patients for hydrocolonic sonography examinations.

Figure – III-7



Specially prepared can and stand for instilling water into  
colon through anus

### III-7: X-RAY INSTRUMENT

Conventional radiography:

X-rays are part of the electromagnetic spectrum. They are used for all conventional radiography. Radiographic images depend on the fact that x-rays are absorbed to a variable extent as they pass through the body. The visibility of the structures and disease depends on this differential absorption. Radiographic contrast agents are used to visualise structures or disease processes that would otherwise be invisible. In double contrast barium enema x-ray barium sulphate and air is used as contrast agents. In this study Diagnostic X-ray unit F78, People's Republic of China, was used. Specifications: Rotating anode x-ray tube XD51-20-40/125. Radiography: Focal spot 2.0x2.0mm, 300 mA, tube voltage 90-125 kv. Fluoroscopy: Focal spot 1.0x1.0mm, 0.5-5 mA, 5 kv in continuous imaging [Fig – III-8].

Figure – III-8



X-ray units for double contrast barium enema x-ray

**METHODS**  

---

**CHAPTER 4**

## **IV: METHODS**

### **IV-1: HYDROCOLONIC SONOGRAPHY:**

#### **Colonic Preparations:**

Patients were instructed to eat an exclusively low-residue diet during the previous day of examination. Two laxative tablets [Tab. Laxenna] were advised to take at night. On the next morning, after bowel moved, the patient came to the centre in empty stomach. At the centre, enema simplex with warm soap-water was given. Usually 1-2 enemas were sufficient to clean the entire colon. Some time more than 2 enemas were needed. The procedures were well tolerated by the patients. No significant adverse effects were noticed during or after colon cleaning.

#### **Hydrocolonosonography:**

A total up to 1500 ml of water [kept two days in a container to make it bubble free] was instilled into colon through anus using an specially prepared big sized enema can and a stand [Fig – IV-1]. Slow instillation of water was done to avoid sudden discomfort of the patient. Continuous abdominal sonographic examination of the colon from the time of water

instillation was carried out with real-time scanning device [Aloka SSD 500 with 5.0 MHz & 3.5 MHz Curvilinear Transducers of Aloka Co. Ltd., Tokyo, Japan. Aloka SSD 1100 with 5.0 MHz Curvilinear and 7.5 MHz Linear Transducers of Aloka Co. Ltd., Tokyo, Japan. Kontron Sigma 21 with 3.5 MHz Sector & 5.0 Curvilinear & 7.5 MHz Biplaner Transrectal Transducers of Kontron Instruments S. A., Montigny Le Bretonneux, France]. No buscopan or any spasmolytics were used.

**Scanning Procedure:**

Abdomen and pelvic area of the patient were well exposed. Sufficient amount of ultrasound gel was applied on the skin of colon area. Examination began as soon as the water began to instill into colon through anus. Rolling or tilting of patient were not necessary. The examination began at rectosigmoid area and gradually covered descending colon, splenic flexure, transverse colon, hepatic flexure, ascending colon, caecum and terminal part of colon. The colon lumen, contents, wall and surrounding areas were evaluated. Usually 15 to 20 minutes were sufficient to complete the examinations. Presence of small amount of faeces did not interfere with real-time examination of the colon and easily could be differentiated from colon mass. If excessive amount of faecal matters were retained in the colon, patients were sent for another enema.



**Pictures:**

Sufficient number of pictures was taken to document the normal and abnormal areas of colon. Sony and Fuji Video Printers using heat sensitive papers were applied for this purpose.

**Other Examinations modalities:**

All the patients were examined with Double Contrast Barium Enema X-ray. Some of the patients were examined by Colonoscope. Some of the patients with colonic pathology were operated and histopathological confirmations were obtained. Ultrasonography examinations of the operated specimens of normal and abnormal colon were done to correlate in vitro images with in vivo images.

Figure – IV-1



Hydrocolonic sonography: Water was instilled into colon through anus using an especially prepared big sized enema can and a stand. Continuous abdominal sonographic examination of colon was done.

## **IV-2: HYDROCOLONOSONOGRAPHIC DIAGNOSIS OF NORMAL AND ABNORMAL COLON:**

### **General Considerations:**

Based on findings of hydrocolonic sonography, the scans were classified as normal or abnormal. In abnormal scans, attempts were made to diagnose the nature of pathology. The sonographic evaluation of inflammatory and neoplastic diseases of the large bowel should include measuring the width of the intestinal lumen and evaluating the appearance of haustra. The sonographic appearance and width of the intestinal wall should be noted along with evidence of localized or disseminated wall thickening or the presence of intraluminal tumors.

By using real time equipment it is possible to observe peristalsis of the fluid filled bowel as well as movement of particulate material or gas bubbles within the bowel lumen. A quick, light compression with the transducer on the abdomen may induce peristalsis and cause stool particles or gas bubbles to move, thereby allowing a rapid differentiation between artifacts and real pathologic alterations of the large bowel wall [Fig – IV-2].

The following criteria were found for normal and abnormal colon in hydrocolonic sonography (Limberg & Osswald 1994, Limberg 1992, Limberg 1990 a, Limberg 1990 b, Limberg 1989, Limberg 1988, Limberg

1987, Limberg 1999, walter et al 1992, Elewaut et al 1995, Hernandez-Socorro et al 1995, Hiroki et al 1989, Bala et al 1999, Bala et al 2000, Bala et al 2001).

**Normal colon:**

The sonographic views obtained using hydrocolonic sonography show the echo-free intestinal lumen with a width of 4 to 5cm, with the haustra appearing as echogenic projections from the bowel wall [Fig – IV-3, IV-4, IV-5 & IV-6]. In most patients the Bauhin's valve is seen as an echogenic polypoid lesion projecting in to the lumen [Fig – IV-7]. Under optimal examination conditions, the junction of the ileum with the caecum can be demonstrated. Using a high-frequency transducer, it is not only possible to judge the haustra and the width of the intestinal lumen, but also view the detailed structure of the intestinal wall [Fig – IV-8]. Five layers can be sonographically distinguished within the wall of the large intestine, along the margin of the lumen a thin echogenic layer is seen. Deep to this there is a thin echo-poor layer followed by a thicker echogenic layer, then a thicker echo-poor layer, and finally an outer echogenic layer [Fig – IV-9]. The total width of the intestinal wall is 4mm. It has been postulated that the first echogenic layer is the surface of the glandular layer of the mucosa, followed by the hypoechoic layer, which corresponds to the glandular layer of the

400508

mucosa plus muscularis mucosa, these two layers together therefore represent the mucosa. The third echogenic layer is the submucosa; the muscularis propria corresponds to the fourth echo-poor layer, and the subserosa and serosa to the fifth echogenic layer. The same type of finding is found in endoluminal ultrasound of gastrointestinal tract [Fig – IV-10] Using colonic sonography, the diagnosis of inflammatory and neoplastic diseases of the large bowel are possible.

#### **Colon polyp:**

Colonic polyps and carcinomas appear sonographically as echogenic structures projecting from the intestinal wall into the lumen after extrinsic compression or a change in patient position. Small polyps cannot always be visualized [Fig – IV-11]; however, polyps larger than 7mm can be identified [Fig – IV-12 & IV-13]. The difficulty in demonstrating small colonic polyps is due to the fact that a definite differentiation between the polyps and nearby haustra is not always possible.

#### **Colon carcinoma:**

Colonic carcinomas appear as rounded echogenic structures that project into the lumen. In contrast to polyps, these have no observable movement in the intestinal lumen; favorable examination conditions allow sonographic demonstration colon wall infiltration as well as changes in the normal layers

[Fig – IV-14]. Sometimes the cancer involves the whole circumference giving the appearance of annular lesion [Fig – IV-15 & IV-16].

### **Crohn's disease:**

Using colonic sonography it is possible to demonstrate acute inflammatory large bowel diseases, e.g., active Crohn's disease and acute ulcerative colitis.

Visualizing the sonographic structure of the intestinal wall is important in differentiating the acute severe inflammatory diseases from each other with colonic sonography. In acute severe colonic Crohn's disease, due to transmural inflammation, the normal stratified appearance of the colonic wall is no longer in evidence and the wall appears visibly thickened [Fig – IV-17]. The colonic wall thickness increases to  $10 \pm 3\text{mm}$  and becomes echo-poor and haustra are no longer visible [Fig – IV-18]. Furthermore, the involved segments of colon are rigid on real-time examination and peristalsis is absent. In addition to demonstrating diffuse inflammatory changes, colonic sonography can also locate intestinal stenosis.

### **Ulcerative colitis:**

Patients with active ulcerative colitis without extensive inflammatory pseudopolyposis will maintain the normal sonographic stratified appearance of the colonic wall. The wall is hypoechoic and is only moderately increased in thickness ( $6 \pm 2\text{mm}$ ). The quality with which colonic sonography reveals

the two layers closest to the lumen that represent the mucosa is remarkable [Fig – IV-19]. As in Crohn's disease, the haustra are no longer visible. In ulcerative colitis with extensive inflammatory pseudopolyposis, the thickness of the gastrointestinal wall increases up to 15mm and the typical stratified appearance of the colonic wall is no longer visible (Fig. 19). In these patients, a clear distinction between active Crohn's disease and ulcerative colitis is difficult and is sometimes impossible. The sonographic detection of extensive inflammatory pseudopolyposis helps in making the diagnosis of acute ulcerative colitis [Fig – IV-20].

Differentiation between Crohn's disease and ulcerative colitis is based on the identification of wall layers. In Crohn's disease all the wall layers are involved and can not be identified separately. But in ulcerative colitis mainly superficial layers are involved and wall layers can be differentiated [Fig – IV-21].

Figure – IV-2



Small amount of fecal material does not interfere with the examination. By real time equipment it is possible to observe the movements of the particulate material or gas bubbles within the bowel lumen.



Figure -- IV-3



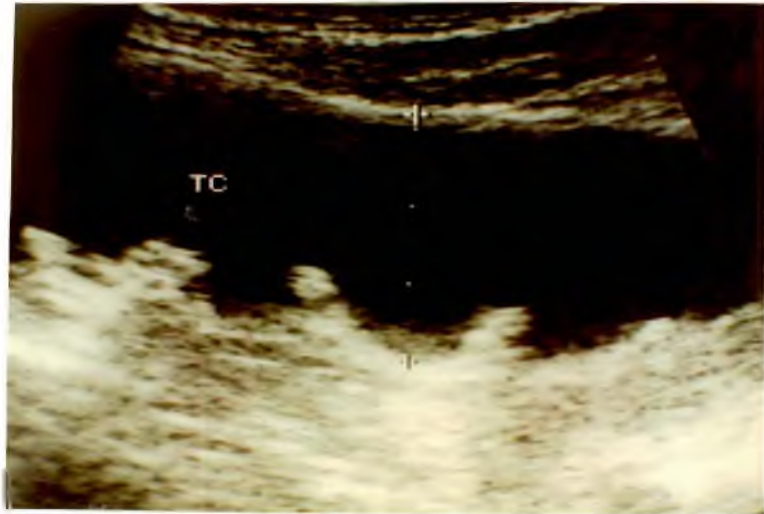
Hydrocolonic sonography showing the echo-free intestinal lumen with a width of 3-5cm and haustra appearing as ecogenic projections from the bowel wall.

Figure – IV-4



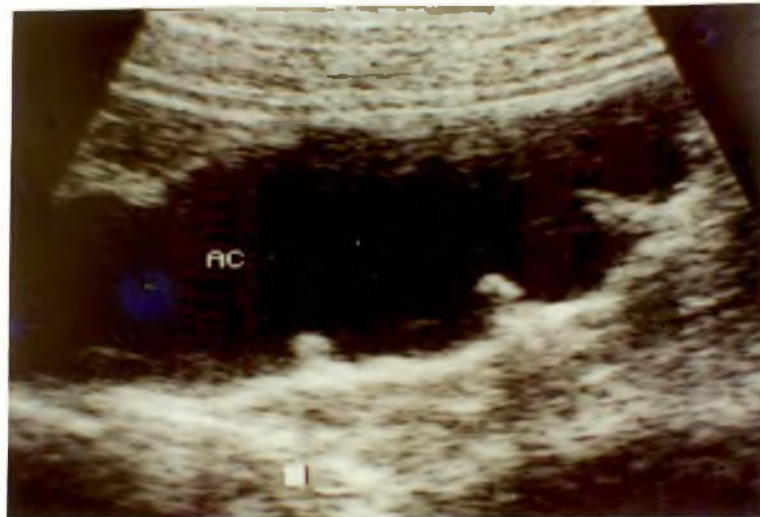
Hydrocolonoscopic sonography of normal descending colon.

Figure – IV-5



Hydrocolonic sonography of normal transverse colon.

Figure – IV-6



Hydrocolonic sonography of normal ascending colon.

Figure – IV-6



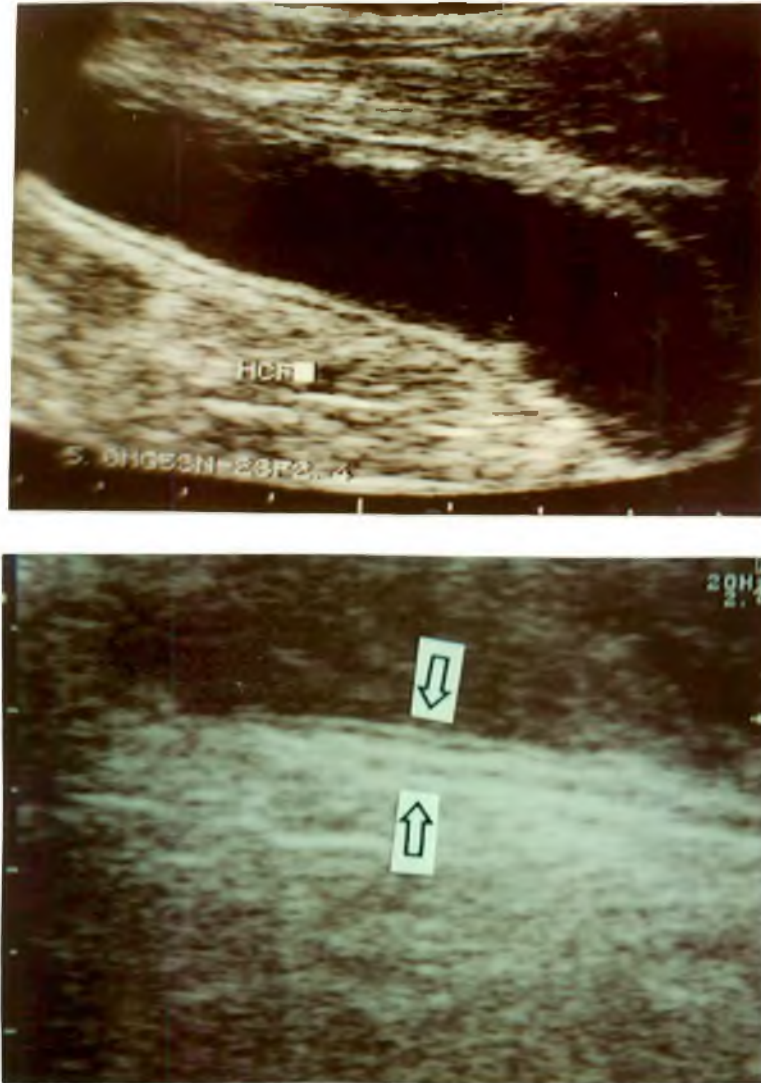
Hydrocolonic sonography of normal ascending colon.

Figure – IV-7



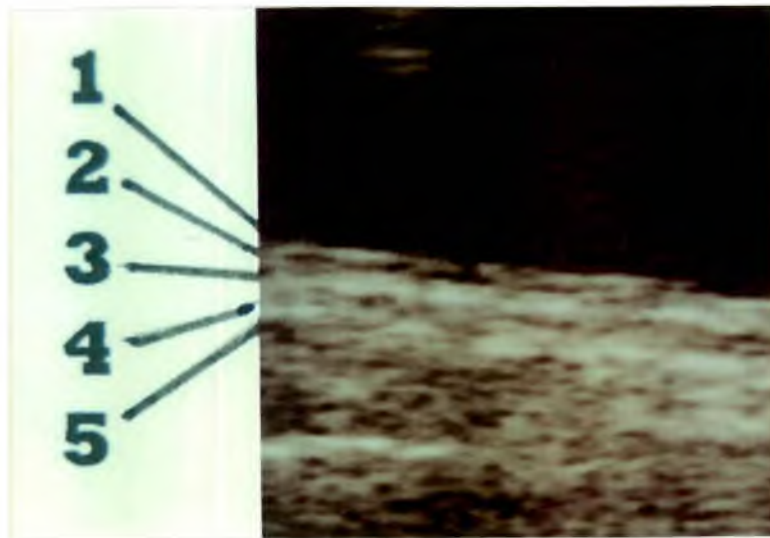
Bauhin's valve is seen as an echogenic polypoid structure projecting into the lumen. In optimal conditions, the junction of the ileum with caecum can be demonstrated.

Figure – IV-8



Using high frequency transducer, the detailed structure of the colon wall can be seen. 5 layers are more evident when 7.5 MHz transducer is used.

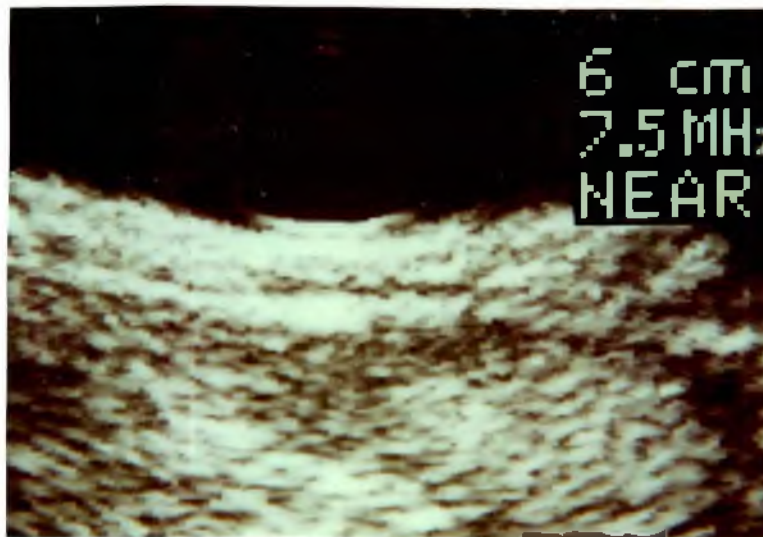
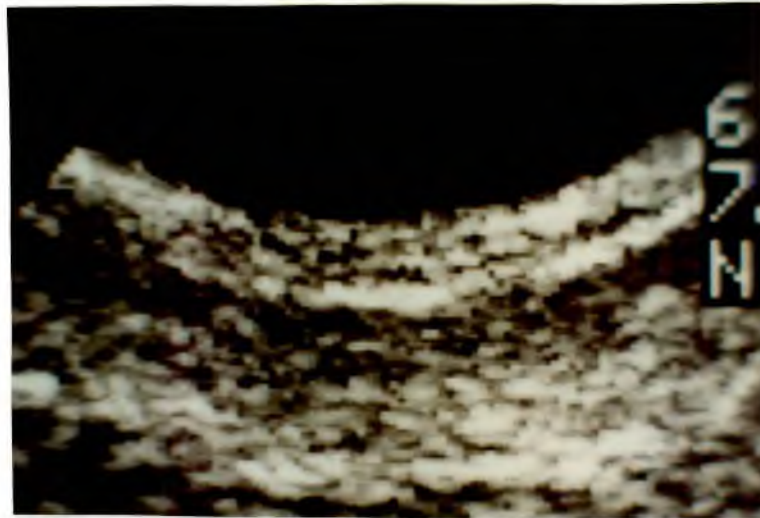
Figure – IV-9



In hydrocolonic sonography five layers can be sonographically distinguished within colon wall: (1) along the margine of the lumen a thin echogenic layer, (2) deep to it is a thin echo-poor layer (3) followed by a thicker echogenic layer, (4) then a thicker echo-poor layer, (5) and finally an outer echogenic layer.



Figure – IV-10



The same type of 5 layers of gastrointestinal tract wall is found in endoluminal ultrasonography. The figures showing 5 layers of rectal wall in transrectal ultrasonography.

Figure -- IV-11



Small colonic polyps appear as echogenic structures projecting from intestinal wall into the lumen.

Figure – IV-12



Polyps larger than 7 mm can be identified easily. Polyps are originated from mucous layer and do not invade deeper layers

Figure – IV-13



Villous adenoma is a benign sessile tumour showing a sponge like appearance.

Figure – IV-14



Carcinoma of colon may allow sonographic demonstration of colon wall infiltration as well as changes in the normal layers.

Figure – IV-15



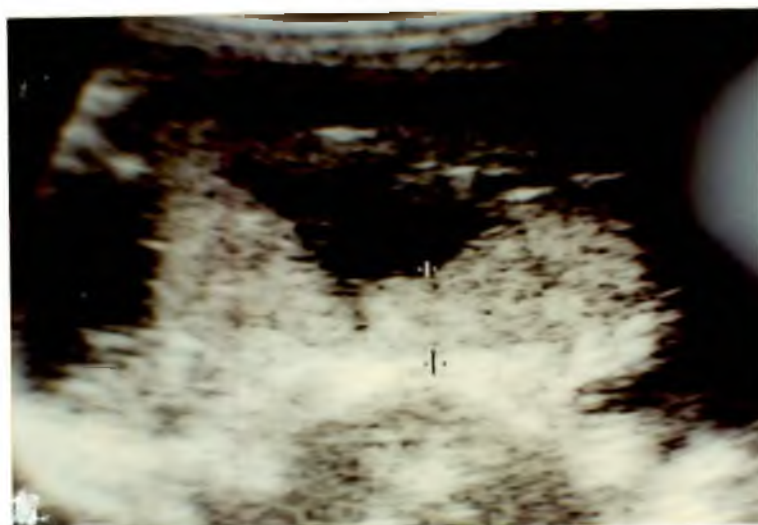
Colon carcinoma may involve whole circumference giving the appearance of annular lesion.

Figure – IV-18



The colonic wall thickness may increase to  $10 \pm 3$ mm and becomes echo-poor and haustra are no longer visible.

Figure – IV-16



Usually carcinoma at the rectosigmoid area involves the whole circumference and giving the appearance of annular lesion

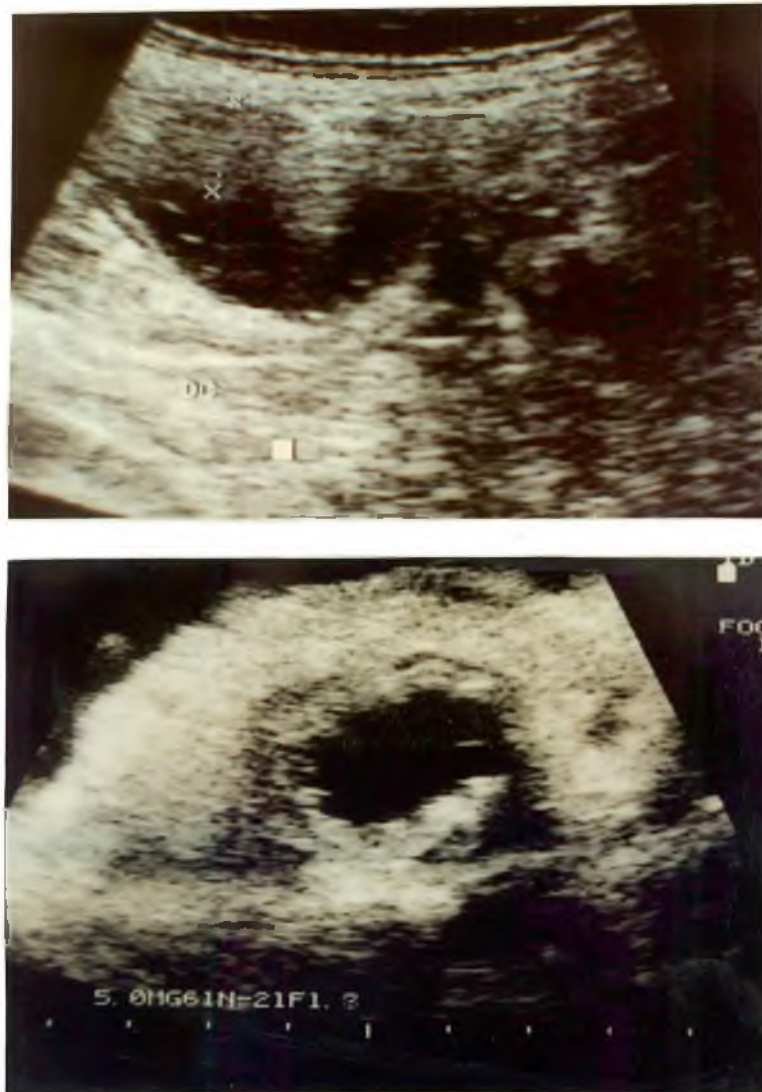


Figure – IV-17



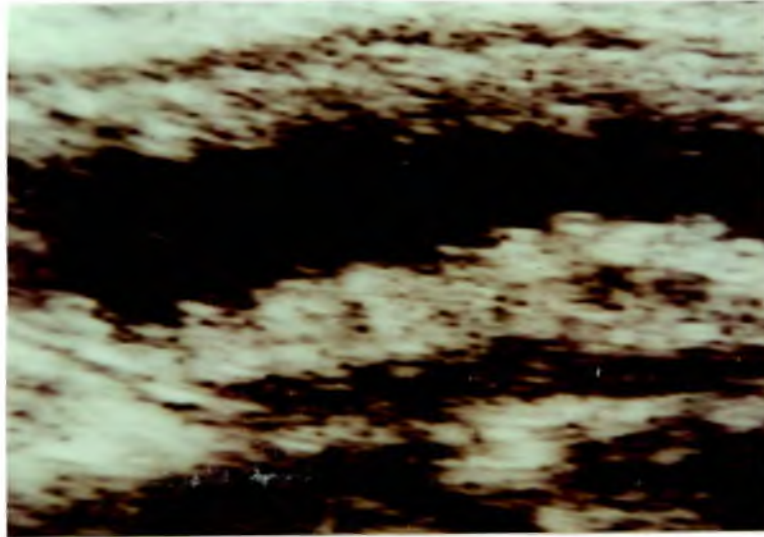
In Crohn's disease, the normal stratified appearance of the colonic wall is no longer evident and the wall appears visibly thickened.

Figure – IV-18



The colonic wall thickness may increase to  $10 \pm 3$ mm and becomes echo-poor and haustra are no longer visible.

Figure – IV-19



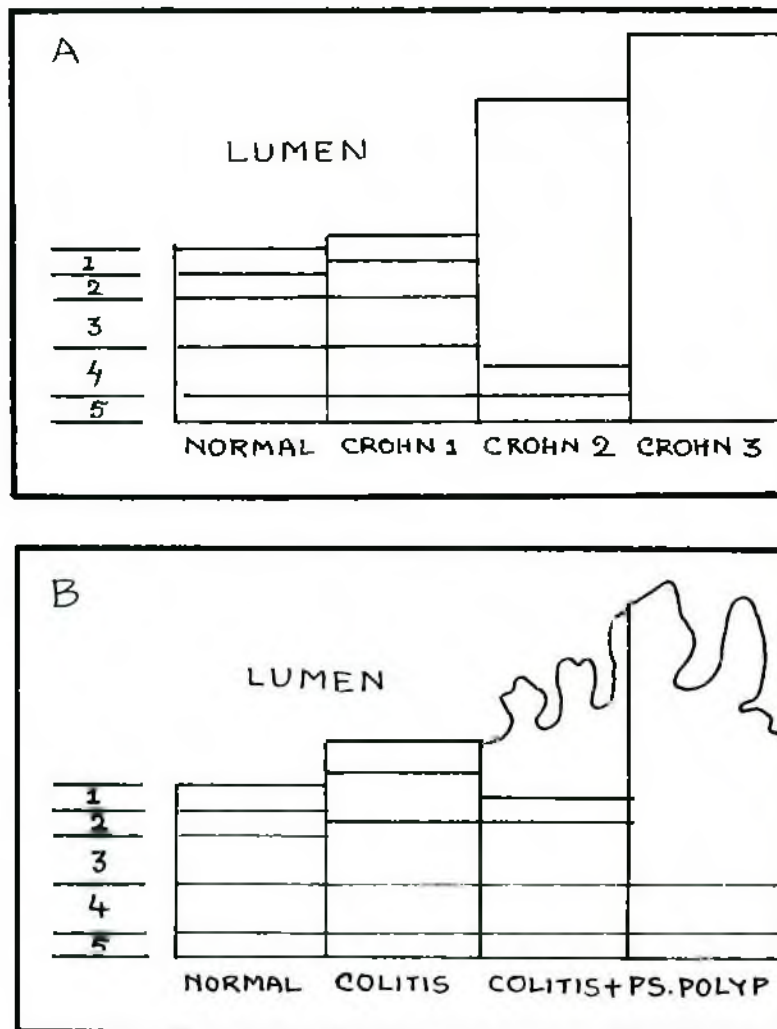
In ulcerative colitis the two layers closest to the lumen that represent the mucosa is remarkably thickened.

Figure – IV-20



The sonographic detection of extensive inflammatory pseudopolyposis helps in making the diagnosis of acute ulcerative colitis.

Figure – IV-21



- A. Sonographic features of Crohn's disease: normal wall with distinct 5 layers. Crohn 1, the colon wall is less echogenic, 5 layers are evident. Crohn 2, colon wall is thickened and hypoechoic, 3 layers not distinguishable. Crohn 3, colon wall is clearly thickened, wall layers are not distinguishable.
- B. Sonographic features of ulcerative colitis. Acute ulcerative colitis, colon slightly thickened, all layers are differentiated. Colitis with pseudopolyps, colon wall is thickened and multiple pseudopolyps are seen.

### **IV-3: CONVENTIONAL ABDOMINAL SONOGRAPHY:**

#### **General Considerations:**

To set the normal and abnormal appearances of colon, conventional abdominal sonography were done frequently. Normal and abnormal colon, small intestine, duodenum and stomach were included in the examinations. 3.5 MHz and 5.0 MHz transducers were used.

#### **Sonographic features of normal bowel:**

Under normal physiological conditions, the wall of the stomach and the esophagogastric junction can be easily discerned during routine ultrasonic evaluation of the upper abdomen. Distal to the duodenum, the normal gastrointestinal tract cannot be routinely evaluated because of gas within loops of bowel, which prevents the transmission of sound waves. On real-time ultrasonic examination, normal bowel segments demonstrate a “target” configuration composed of a strong echogenic center surrounded by a sonolucent rim corresponds to the bowel wall [Fig – IV-22]. The use of a high frequency transducer enables a more detailed evaluation of the antral wall [Fig – IV-23], especially when fluid is present in the stomach [Fig – IV-24]. The anechoic rim is caused by the muscle layer of the gastrointestinal wall and the echogenic layer situated lumenally is created by the gastric mucosa. The fluid in the stomach is represented by the hypoechoic center of

the “target”. The thickness of the normal gastric body and antral wall can measurement remains fairly constant over its entire length. A change in the overall configuration of the target can be induced by compression with the transducer on the abdomen that generates peristaltic waves clearly visible with the use of real-time ultrasonic equipment.

Sonographic features of the bowel wall in inflammatory and tumorous diseases. Pathologic involvement of a bowel segment results in concentric or eccentric thickening of the bowel wall. A bowel wall thickness greater than 5mm should be considered pathologic. The thickening of the bowel wall produces a “target” appearance on transverse section and a “pseudokidney” [Fig – IV-25] shaped appearance on longitudinal section. These, infiltrated segments of bowel are stiff and shows no peristalsis or movement of the internal contents. Compression with the transducer does not induce a change in the overall configuration of the lesion. Demonstration of a lesion with a target or pseudokidney appearance distal to the duodenum should be considered pathologic. Although the target pattern was first described in cases of malignant tumors, it is now recognized that it can be found in both tumorous and inflammatory infiltration of the gastrointestinal wall. It can be observed in a variety of benign intestinal diseases including Crohn’s disease ulcerative colitis, ileocolic intussusception, and pyloric stenosis. The

only a single target lesion is demonstrated. The clinical relevance of the target sign as an indication of disease of the large intestine is further hampered by the high percentage of false positive findings. Also, colonic polyps cannot be detected by transabdominal sonography and colonic carcinomas can only be recognized when in an advanced stage [Fig – IV-26].



Figure – IV-22



Normal bowel segments demonstrate a 'target' configuration composed of a strong echogenic centre surrounded by a sonolucent rim correspond to bowel wall.

Figure – IV-23



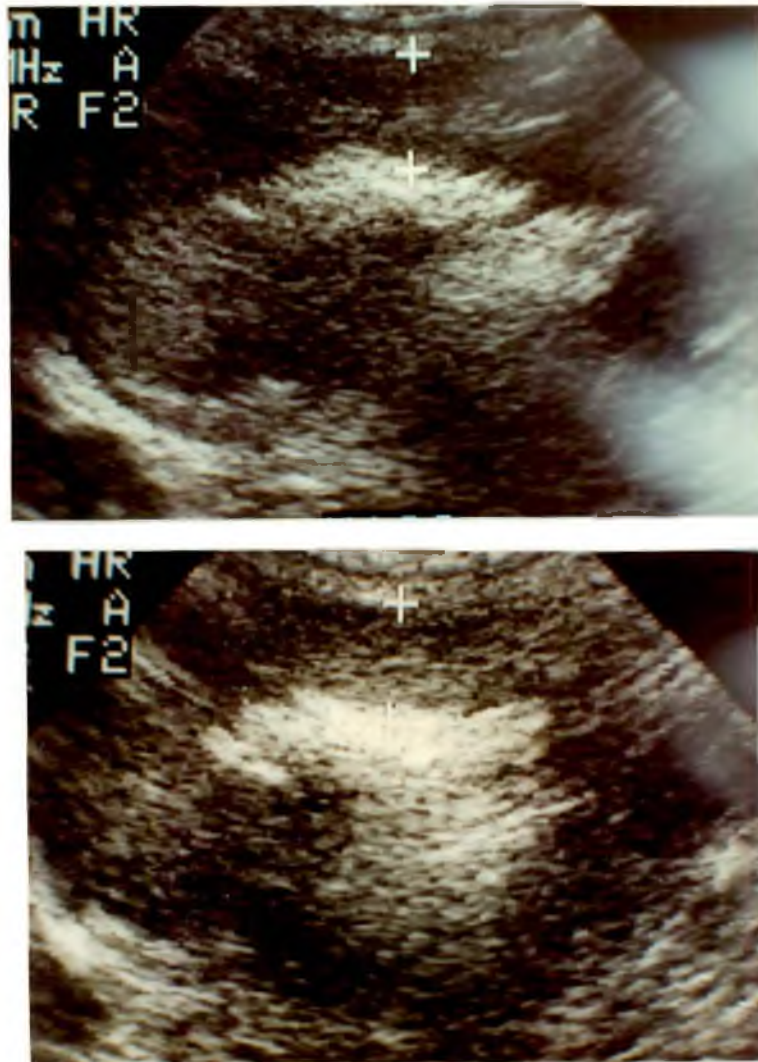
The use of high frequency transducer enables a more detail evaluation of the antral wall. When fluid is present in the stomach the wall becomes more evident.

Figure – IV-24



5 layers of the gastrointestinal wall can be identified by conventional ultrasonography examinations if high frequency transducer is used.

Figure – IV-25



The thickening of bowel wall produces a target appearance on transverse section and a pseudokidney shaped appearance on longitudinal section

Figure – IV-26



By transabdominal ultrasonography, colonic carcinomas can only be recognized when in an advanced stage

#### **IV-4: IN VITRO ULTRASOUND EXAMINATION OF COLON:**

##### **Water bath examination:**

In an attempt to evaluate the ultrasound appearance *in vitro*, eight normal colon resection specimens, five specimens of colon carcinoma [Fig – IV-27], two specimens of colon polyp one ulcerative colitis and one Crohn's disease were examined.

The controls were tumor-free resections of patients with colonic tumors. The specimens were fixed in 10% formaldehyde solution and examined in a water bath with a 5-MHz transducer [Aloka SSD 500 & Aloka SSD 1100] [Fig – IV-28]. In patients with diseases, biopsies were taken from those intestinal sections that had a normal sonographic appearance and from those areas that showed the most prominent sonographic changes [Fig – IV-29]. The histological findings were compared with the sonographic findings. In addition, those patients with colon mass received a preoperative colonic sonography examination. After a retrograde instillation the images of *in vitro* examinations were compared with *in vivo* images.

In order to compare the *in vivo* and *in vitro* appearances of colon, identical segments of colon were selected. The specific points selected for comparison were localized *in vivo* and *in vitro*. The length and the wall thickness of the pathological segments were measured by electronic callipers. The findings

assessed in vitro on the resections were then compared with the sonographic changes demonstrated in vivo by colonic sonography. For diagnosis of colonic disease, sonographic criteria included the thickness of the colon wall, its echogenicity, and the obliteration of the five-layer wall stratification.

Figure – IV-27



Surgically resected specimens of colon were examined



Figure – IV-28



Ultrasonographic examination of colon specimen was done  
by placing it in water bath

Figure – IV-29



Ultrasound image of a colon specimen

#### **IV-5: DOUBLE CONTRAST BARIUM ENEMA X-RAY:**

##### **General Considerations:**

The barium enema is the routine method of examination of colon. In 1950s double-contrast study was introduced. Over the past three decades the superiority of the double-contrast method has become widely established.

Adequate preliminary preparation of the colon to remove fecal material prior to the administration of barium enema is vital. In this study, double-contrast barium enema x-ray [BEX] was done with the same preparation given to the patients for hydrocolonic sonography. This involved low residual diet in the previous day with two laxative tablets [Tab. Laxenna] at night. On the day of examination patient was given enema simplex with warm soap water. One or two enemas were sufficient. After examining by ultrasound the patient was sent for BEX examination. The preparation for hydrocolonic sonography was found sufficient for BEX examination. If the patient was examined at a different day, a separate preparation was given.

A barium sulphate and water mixture was administered through a rectal catheter by gravity. Barium was run in as far as the mid-transverse colon, at which stage the tube was clamped and rectum drained. Air was then insufflated, with rotation of patient as necessary, until the whole colon is

coated with barium and air. Required number of films was taken to evaluate the whole colon.

**Normal colon:**

The radiological anatomy of normal colon is shown in [Fig – IV-30]. The length of the colon is very variable. The caliber decreases from the caecum to sigmoid colon.

The caecum is usually situated in the right iliac fossa, but the position is variable. The lips of the ilio-caecal valve may project into the caecum and cause a filling defect. Filling of terminal ileum and appendix may occur in some patients [Fig – IV-31].

Haustra can usually be recognized in the whole of the colon although they may be absent in the descending and sigmoid regions. The outline of the distended colon is smooth.

**Ulcerative colitis:**

Ulcerative colitis is a disease of unknown etiology characterized by inflammation and ulceration of colon. The disease always involves the rectum. When more extensive it extends in continuity around the colon, sometimes affecting the whole colon. The cardinal radiological sign is widespread ulceration. The ulcers are usually shallow but in severe cases may be quite deep. There is loss of normal colonic haustra in the affected

portion of the colon. Narrowing and shortening of the colon, giving the appearance of a rigid tube, and pseudopolyps are seen in advanced disease. Pseudopolyps are small filling defects projecting into the lumen.

When the whole colon is involved, the terminal ileum may become dilated. Toxic dilatation is a serious complication and barium enema should not be done.

**Crohn's disease:**

Crohn's disease is a chronic granulomatous condition of unknown etiology, which may affect any part of the gastrointestinal tract, but most frequently involves the lower ileum and colon.

At an early stage in the disease, the findings at barium enema are: loss of haustration, narrowing of the lumen of the bowel and shallow ulceration [Fig – IV-32]. This criss-cross ulceration combined with mucosal edema may give rise to a 'cobblestone' appearance. Later, the ulcers become deeper.

Strictures are a common finding in Crohn's disease. The strictures are smooth and have tapered ends. The disease is not always circumferential. Areas of disease with intervening normal bowel may occur. The rectum is often spared.

### **Diverticular disease:**

Diverticula are sac-like out-pouchings of mucosa through the muscular layer of the bowel wall. They are associated with hypertrophy of the muscle layer and are probably due to herniation of mucosa through areas of weakness where blood vessels penetrate the muscle. Diverticula are very common, particularly in the elderly. They are seen all part of colon but is commonest in the sigmoid colon.

The diverticula when filled with barium are seen as spherical out-pouchings with a narrow neck. The colon may also show a 'saw tooth' serrated appearance due to hypertrophy of the muscle coats.

A diverticulum may perforate, resulting in a pericolic abscess or fistula into the bladder, small bowel or vagina. A stricture with or without local abscess formation may occur.

### **Tuberculosis:**

Tuberculosis of the colon commonly involves the ileocaecal region, producing rigidity, shortening and distortion of the caecum. Marked thickening of the bowel wall occurs due to a combination of granulomatous infiltration, caseation and fibrosis. As a result the bowel lumen becomes considerably narrowed with associated mucosal irregularity. Ulceration, when it occurs, tends to be circumferential. Tuberculosis of the more distal

colon tends to produce segmental narrowing or stricture formation of varying length.

**Polyps:**

The word 'polyp' means a small mass of tissue arising from the wall of the bowel projecting into the lumen. Polyps may be sessile or on a stalk, single or multiple. Polyps may be neoplastic, inflammatory or occasionally developmental in origin.

Villous adenoma is a benign sessile tumor showing a sponge-like appearance due to barium trapped between the villous strands [Fig – IV-33]. In familial polyposis numerous small polyps are present throughout the colon [Fig – IV-34].

It is often impossible on radiological grounds to exclude malignancy in a polyp. However, a tiny minority of polyps less than 1 cm in size and very few less 2cm are cancers. The features that suggest malignancy are: a diameter of more than 2cm, a short thick stalk, irregular surface, rapid rate of growth as judged by serial barium enema examinations.

**Carcinoma:**

Carcinomas may arise anywhere in the colon but they are commonest in the rectosigmoid region and the caecum. The appearance and behaviour of a carcinoma in these two sites are usually quite different. The patient with a

rectosigmoid carcinoma often has an annular stricture and presents with alteration in bowel habit and obstruction [Fig – IV-35], whereas with a caecal carcinoma the tumor can become very large without obstructing the bowel, so anemia and weight loss are the common presenting features.

A barium enema shows the annular carcinoma as an irregular stricture with shouldered edge [Fig – IV-36], such strictures are more than 6cm in length. The polypoid or fungating carcinoma causes an irregular filling defect projecting into the lumen of the bowel.

Multiple primary tumors must be excluded, as the patient with one carcinoma of the colon has a higher than normal risk of developing a second colonic cancer. This may be present at the time of the diagnosis or may present after the first tumor has been removed. Spot films are suitable for diagnostic purpose [Fig – IV-37].



Figure – IV-30



The radiological anatomy of normal colon as shown in the image of double contrast barium enema x-ray

Figure – IV-31



In barium enema x-ray, filling of terminal ileum and appendix may occur.

Figure – IV-32



At an early stage of Crohn's disease, findings at barium enema are: loss of haustration, narrowing of lumen and shallow ulceration.

Figure – IV-33



Benign sessile tumour showed a sponge-like appearance due to barium trapped between villous strands

Figure – IV-34



In familial polyposis numerous small polyps are present throughout the colon

Figure – IV-35



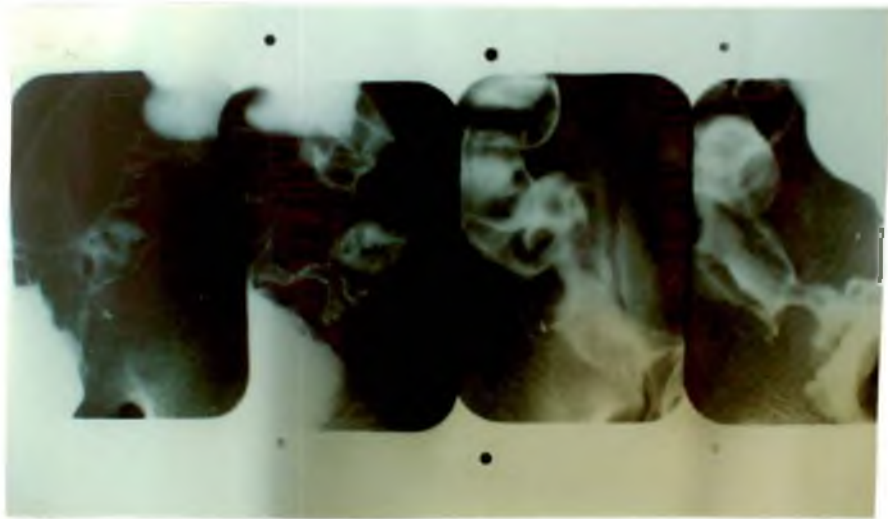
Rectosigmoid carcinoma has an annular stricture.

Figure – IV-36



A barium enema shows the annular carcinoma as an irregular stricture with shouldered edge.

Figure – IV-37



A barium enema spot films can diagnose pathologies more accurately



#### **IV-6: HISTOPATHOLOGICAL DIAGNOSIS OF COLON DISEASES:**

##### **Normal colon:**

The large intestine consists of a mucosal membrane with no folds except in its distal [rectal] portion. No villi are present in this portion of the intestine.

The intestinal glands are long and characterized a great abundance of goblet and absorptive cells and a small number of enteroendocrine cells. The absorptive cells are columnar and have short, irregular microvilli.

The lamina propria is rich in lymphoid cells and in nodules that frequently extend into the submucosa. The richness in lymphoid tissue is related to abundant bacterial population of the largeintestine. The muscularis comprises longitudinal and circular strands. This layer differs from that of the small intestine, because fibres of the outer longitudinal layer congregate in three thick bands called tenia coli. In the intraperitoneal postions of the colon, the serous layer is characterized by small, pendulous protuberances composed of adipose tissue – the appendices epiploicae [Fig – IV-38].

##### **Crohn's Disease:**

Early descriptions of CD emphasized the sharply segmental transmural fibrosis and thickening of the terminal ileum, giving rise to the designation terminal ileitis. Recognition that other sharply delineated small intestinal segments might be affected, with intervening unaffected (“skip”) areas.

In CD, there is gross involvement of the small intestine alone in about 40% of cases, of small intestine and colon in 30%, and of the colon alone in about 30%. CD may involve the duodenum, stomach, esophagus, and even mouth, but these sites are distinctly uncommon. When fully developed, CD is characterized by (1) sharply delimited and typically transmural involvement of the bowel by an inflammatory process with mucosal damage, (2) the presence of noncaseating granulomas in 40% to 60% of cases, and (3) fissuring with formation of fistulae. In diseased segments, the serosa becomes granular and dull gray, and often the mesenteric fat wraps around the bowel surface ("creeping fat"). The intestinal wall is rubbery and thick, the result of edema, inflammation, fibrosis, and hypertrophy of the muscularis propria. As a result, the lumen is almost always narrowed; in the small intestine this is evidenced radiographically as the "string sign," a thin stream of barium passing through the diseased segment. Strictures may occur in the colon but are usually less severe. A classic feature of CD is the sharp demarcation of diseased bowel segments from adjacent uninvolved bowel. When multiple bowel segments are involved, the intervening bowel is essentially normal ("skip" lesions).

By microscopic examination, the mucosa exhibits several characteristic features (Fig - IV-39): (1) epithelial layer and accumulation within crypts to

form crypt abscesses; (2) ulceration, which is the usual outcome of active disease; and (3) chronic mucosal damage in the form of architectural distortion, atrophy, and metaplasia (including rudimentary gastric metaplasia in the intestine). Granulomas are an inconstant finding but may be present anywhere in the alimentary tract, even in patients with CD limited to one bowel segment. Conversely, the absence of granulomas does not preclude a diagnosis of CD. In diseased segments, the muscularis mucosa and muscularis propria are usually markedly thickened and fibrosis affects all tissue layers.

Particularly important in patient with long-standing chronic disease are dysplastic changes appearing in the mucosal epithelial cells. These may be focal or widespread, tend to increase with time, and are thought to be related to a five fold to six fold increased risk of carcinoma, particularly of the colon.

### **Ulcerative Colitis:**

Ulcerative colitis (UC) is an ulceroinflammatory disease affecting the colon but limited to the mucosa and submucosa except in the most severe cases. UC begins in the rectum and extends proximally in a continuous fashion, sometimes involving the entire colon.

With severe disease, there is extensive and broad-based ulceration of the mucosa in the distal colon or throughout its length. Isolated islands of regenerating mucosa bulge upward to create pseudopolyps. Often the undermined edges of adjacent ulcers interconnect to create tunnels covered by tenuous mucosal bridges. The pathologic features of UC are those of mucosal inflammation, ulceration, and chronic mucosal damage. First, a diffuse, predominantly mononuclear inflammatory infiltrate in the lamina propria is almost universally present, even at the time of clinical presentation. Neutrophilic infiltration of the epithelial layer may produce collections of neutrophils in crypt lumina (crypt abscesses). With remission of active disease, granulation tissue fills in the ulcer craters, followed by regeneration of the mucosal epithelium. Submucosal fibrosis and mucosal architectural disarray and atrophy remain as residua of healed disease. The most serious complication of UC is the development of colon carcinoma.

#### **Colonic Tuberculosis:**

Intestinal tuberculosis may be contracted by drinking of contaminated milk. It may be preceded by tuberculous involvement of the lymphoid tissue. Ingested *Mycobacterium tuberculosis* incites chronic inflammation and granulation formation in mucosal lymphoid tissue – particularly Peyer's patches in the terminal ileum, and regional lymph nodes [Fig – IV-40].

### **Colonic Diverticulosis:**

A diverticulum is a blind pouch leading off the alimentary tract, lined by mucosa, that communicates with the lumen of the gut. Congenital diverticula have all three layers of the bowel wall (mucosa, submucosa, and most notably the muscularis propria) and are distinctly uncommon. The prototype is Meckel's diverticulum.

Most colonic diverticula are small, flasklike or spherical outpouchings, usually 0.5 to 1 cm in diameter. They are in the sigmoid colon in approximately 95% of patients. Infrequently, more proximal levels and sometimes the entire colon are affected. Isolated caecal diverticula also occur. The exaggerated peristalsis often induces muscular hypertrophy in affected segments, with unusually prominent taenia coli and circular muscle bundles.

### **Tumors of the Colon:**

Epithelial tumors of the intestines are a major cause of morbidity and mortality worldwide. The colon, including the rectum, is host to more primary neoplasms than any other organ in the body. Colorectal cancer ranks second only to bronchogenic carcinoma among the cancer killers. Adenocarcinomas constitute the vast majority of colorectal cancers and represent 70% of all malignancies arising in the gastrointestinal tract.

### **Non-Neoplastic Polyps:**

The overwhelming majority of intestinal polyps occurs sporadically, particularly in the colon, and increase in frequency with age. Non-neoplastic polyps represent about 90% of all epithelial polyps in the large intestine and are found in more than half of all persons age 60 years or older. Most are hyperplastic polyps, which are small (less than 5mm in diameter), nipple-like, hemispheric, smooth protrusions of the mucosa. They may occur singly but are more often multiple. Although they may be anywhere in the colon, well over half are found in the rectosigmoid region. Histologically, they contain abundant crypts lined by well-differentiated goblet or absorptive epithelial cells, separated by a scant lamina propria [Fig – IV-41].

### **Adenomas:**

Adenomas are neoplastic polyps that range from small, often pedunculated tumors to large lesions that are usually sessile. All adenomatous lesions arise as the result of epithelial proliferation and dysplasia, which may range from mild to so severe as to constitute carcinoma in situ. Furthermore, there is strong evidence that most, and perhaps all, invasive colorectal adenocarcinomas arise in preexisting adenomatous lesions.

Tubular adenomas may arise anywhere in the colon, but about half are found in the rectosigmoid, the proportion increasing with age. In about half of the

instances they occur singly, but in the remainder two or more lesions are distributed at random. Most adenomas have slender stalks 1 to 2cm long and raspberry-like heads and rarely exceed 2.5cm in diameter. Histologically, the stalk is covered by normal colonic mucosa but the head is composed of neoplastic epithelium, forming branching glands lined by tall, hyperchromatic, somewhat disorderly cells, which may or may not show mucin secretion. In some instances there are small foci of villous architecture. In the clearly benign lesion, the branching glands are well separated by lamina propria and the level of dysplasia or cytologic atypia is slight. However, all degrees of dysplasia may be encountered, ranging up to cancer confined to the mucosa (Intramucosal carcinoma) or invasive carcinoma extending into the submucosa of the stalk. A frequent finding in any adenoma is superficial erosion of the epithelium, the result of mechanical trauma.

Villous adenomas are the larger and more ominous of the epithelial polyps. They tend to occur in older persons, most commonly in the rectum and rectosigmoid, but they may be located elsewhere. They generally are sessile, up to 10cm in diameter, velvety or cauliflower-like masses projecting 1 to 3cm above the surrounding normal mucosa. The histology is that of frondlike villiform extensions of the mucosa covered by dysplastic,

sometimes piled-up, columnar epithelium. All degrees of dysplasia may be encountered, and invasive carcinoma is found in up to 40% of these lesions, the frequency being correlated with the size of the polyp.

Tubulovillous adenomas are composed of a broad mix of tubular and villous areas. They are intermediate between the tubular and the villous lesions in their frequency of having a stalk or being sessile, their size, the degree of dysplasia, and the risk of harboring intramucosal or invasive carcinoma.

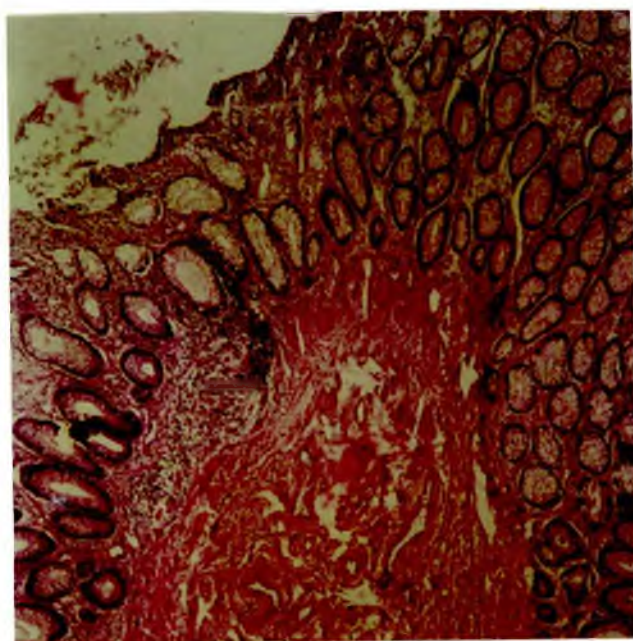
### **Colorectal Carcinoma:**

A great majority (98%), of all cancers in the large intestine are adenocarcinomas. They represent one of the prime challenges to the medical profession, because they almost always arise in adenomatous polyps that are generally curable by resection. With an estimated 150,000 new cases per year, and about 58,000 deaths, this disease accounts for nearly 15% of all cancer-related deaths in the United States. About 25% of carcinomas are in the caecum or ascending colon and a similar proportion in the rectum and distal sigmoid. An additional 25% are in the descending colon and proximal sigmoid; the remainder are scattered elsewhere. No longer are more than half of colorectal cancers readily detectable by digital or proctosigmoidoscopic examination. Most often carcinomas occur singly and have frequently obliterated their adenomatous origins. When multiple

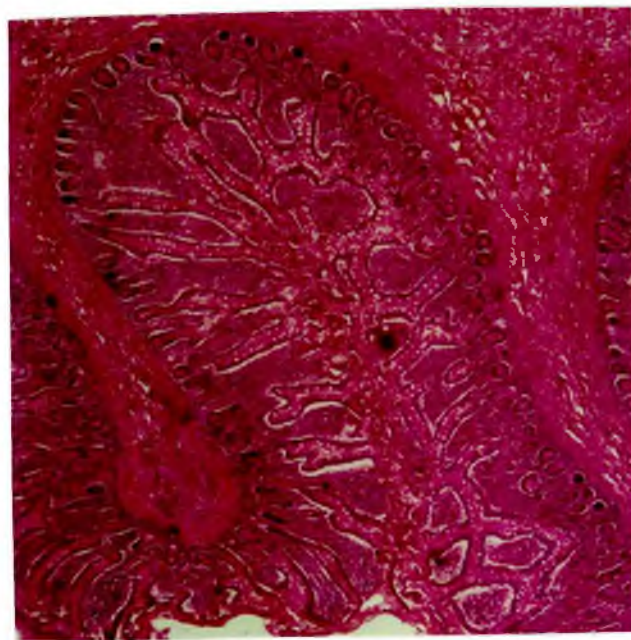
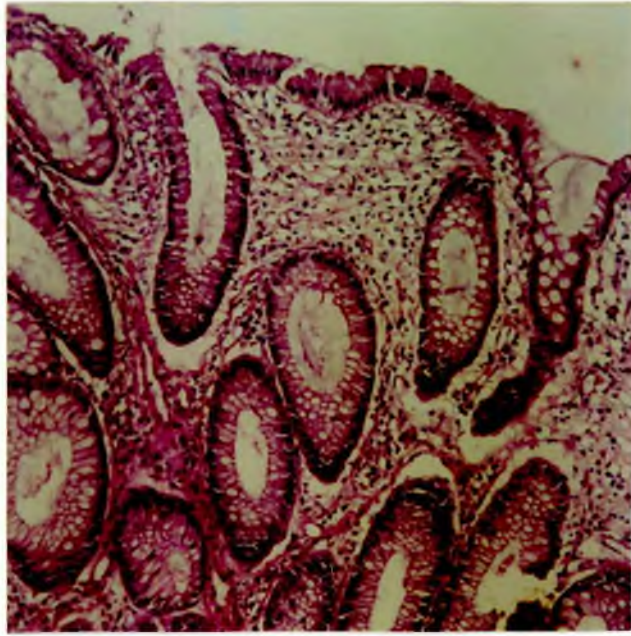


carcinomas are present, they are often at widely disparate sites in the colon. Whereas most cases occur sporadically, 1% to 3% of colorectal carcinomas occur in patients with familial adenomatous polyposis or inflammatory bowel disease. Although all colorectal carcinomas begin as in situ lesions, they evolve into different morphologic patterns. Tumors in the proximal colon tend to grow as polypoid, fungating masses that extend along one wall of the capacious cecum and ascending colon. Obstruction is uncommon. When carcinomas in the distal colon are discovered, they tend to be annular, encircling lesions that produce so-called napkin ring constrictions of the bowel and narrowing of the lumen; the margins of the napkin ring are classically heaped up. Both forms of neoplasm directly penetrate the bowel wall over the course of time (probably years) and may appear as firm masses on the serosal surface. Regardless of their gross appearance, all colon carcinomas are microscopically similar. Almost all are adenocarcinomas that range from well-differentiated to undifferentiated, frankly anaplastic masses. Many tumors produce mucin, which is secreted into the gland lumina or into the interstitium of the gut wall. Because these secretions dissect through the gut wall, they facilitate extension of the cancer and worsen the prognosis [Fig – IV-42].

Figure – IV-38

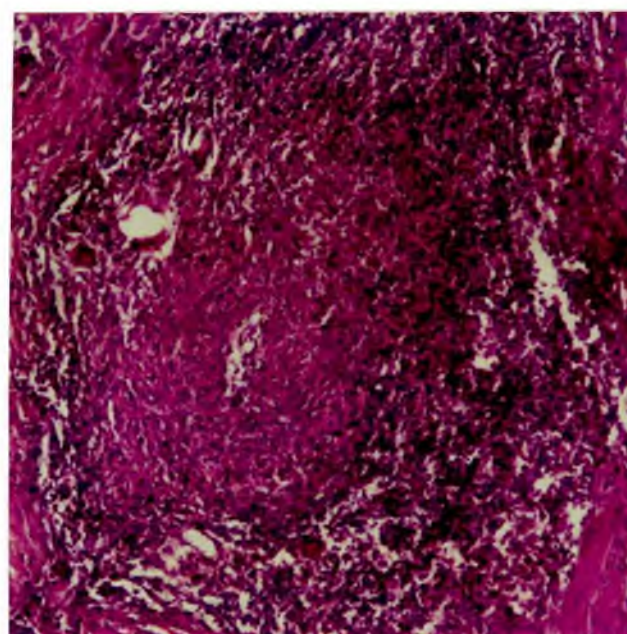
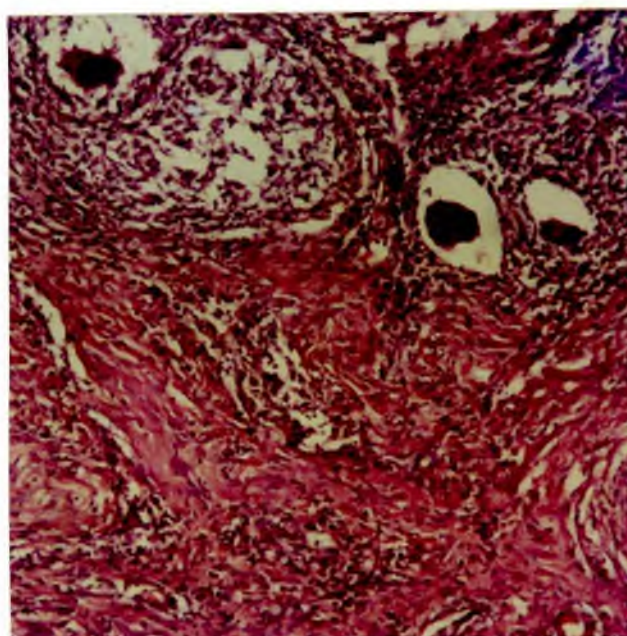


Histopathology of normal colon wall x450



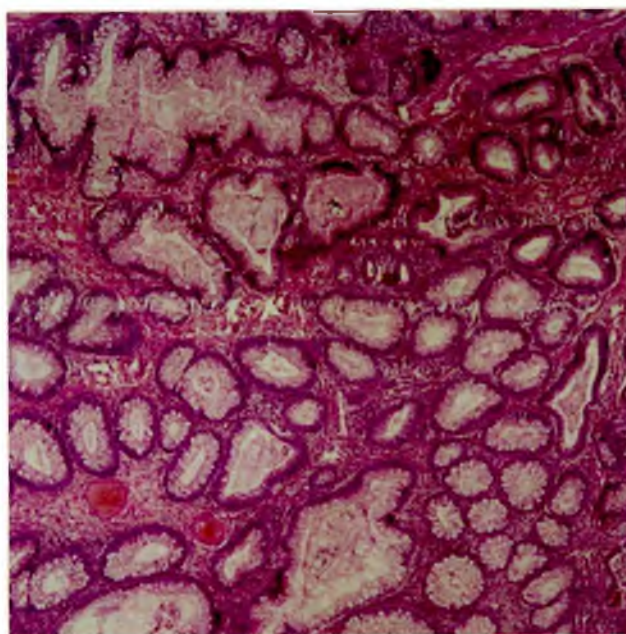
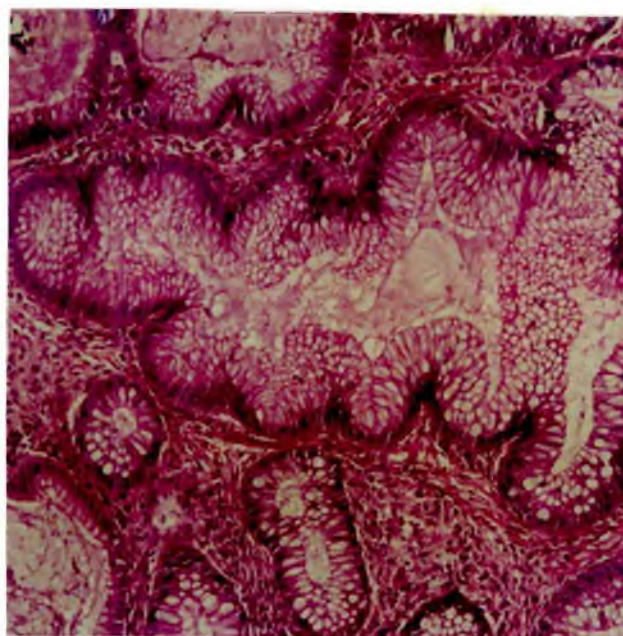
- (a) Crohn's disease showing inflammatory cell in the lamina propria x450,  
(b) Crohn's disease showing submucosal oedema x450

Figure – IV-40



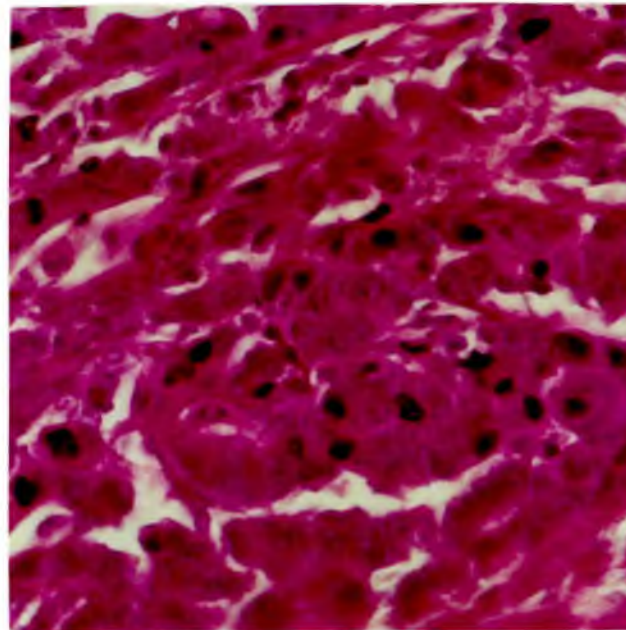
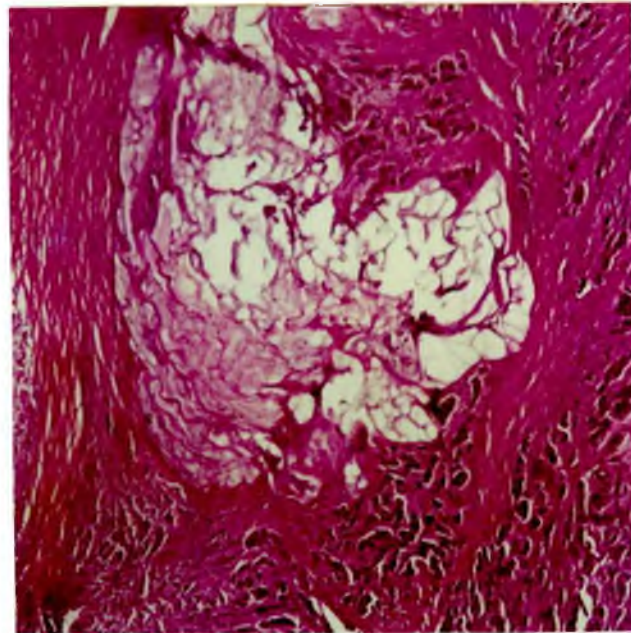
Granulomatous inflammation of colon histologically tuberculosis x150

Figure – IV-41



- (a) Hamartomata Polyp of colon showing dilated glands x450  
(b) Hamartomata Polyp of colon showing dilated glands and inflammatory cells x450

Figure – IV-42



- (a) Carcinoma of colon showing mucin pool within the muscle coat x150  
(b) Poorly differentiated carcinoma showing pleomorphism of cell x450

#### IV-7: METHOD OF CALCULATION:

Nosologic as well as diagnostic sensitivity and specificity of the hydrocolonic sonography diagnoses are calculated according to the following definitions.

The predictive value of the positive hydrocolonic sonography diagnosis and the predictive value of the hydrocolonic sonography diagnosis are calculated accordingly.

95% confidence limits are printed in parenthesis after the percentage alleged.

**1| Nosologic sensitivity =**

$$\frac{\text{Number of true positive hydrocolonic sonography diagnoses}}{\text{Total number of positive final diagnoses}}$$

**2| Nosologic specificity =**

$$\frac{\text{Number of true negative hydrocolonic sonography diagnoses}}{\text{Total number of negative final diagnoses}}$$

**3| Diagnostic sensitivity = Predictive value of negative HS**

$$\frac{\text{Number of true negative hydrocolonic sonography diagnoses}}{\text{Total number of negative hydrocolonic sonography diagnoses}}$$

**4| Diagnostic specificity = Predictive value of positive HS**

$$\frac{\text{Number of true positive hydrocolonic sonography diagnoses}}{\text{Total number of positive ultrasound diagnoses}}$$

**RESULTS**  

---

**CHAPTER 5**



## **V: RESULTS**

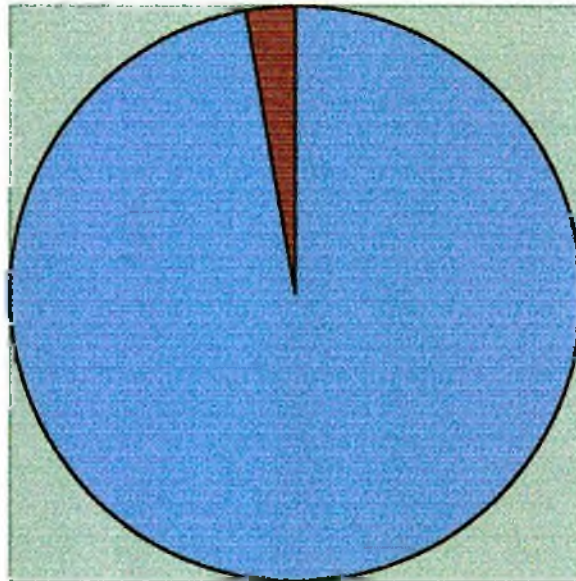
### **V-1: TOTAL PATIENTS:**

Out of 523 patients, in 508 [97.13%] patients it was possible to evaluate the entire length of colon starting at the rectosigmoid junction and ending at the caecum by hydrocolonic sonography. The rectum could not be evaluated due to its location behind the symphysis pubis. In the 15 [2.87%] patients, the examination could not be done completely due to presence of gas in the colon and the patients refused to take further enema for completing the examination [Chart – V-1]. The procedure was well tolerated by each of the patients. There was no failure of examination because of the inability of the patients to maintain a filled colon. There was no need to give hyasomide injection to the patients for relaxation of the bowel. Total examination time was 15-20 minutes.

The patients were classified into Group-I and Group-II according to confirmation of the final diagnosis by colonoscopy and or surgery.

Chart – V-1

**Total Patients = 523**



■ Complete ■ Incomplete □ Slice 3 □ Slice 4

Chart – V-1: Showing out of 523 patients in 508 patients it was possible to examine entire length of colon, while in 15 patients it was not possible to examine entire length of colon

## **V-2: GROUP-I PATIENTS:**

Total 368 patients were included into this group. After proper preparation all the patients were examined by hydrocolonic sonography. Proper hard copy recordings were obtained. After completing the examination, the patients were sent for double contrast barium enema x-ray of colon. X-ray films were obtained for hard copy documentation. Diagnoses were made based on the sonographic and radiographic findings of the examinations respectively. Findings were correlated and presented in Table – V-1.

Out of 368 patients, hydrocolonic sonography [HS] detected 244 [66.30%] patients as normal with all the 5 layers of wall and normal haustra. 38 [10.33%] had carcinoma, 35 [9.51%] polyp, 20 [5.43%] Crohn's disease, 27 [7.34%] ulcerative colitis and 4 [1.09%] had diverticulitis.

On the other hand, out of 368 patients double contrast barium enema x-ray [BEX] detected 252 [68.48%] patients as normal, 36 [9.78%] had carcinoma, 34 [9.24%] polyp, 16 [4.35%] Crohn's disease, 24 [6.52%] ulcerative colitis and 6 [1.63%] had diverticulitis.

Table – V-1

Lesions of colon	Number of detected lesions	
	Hydrocolonic sonography	Double contrast barium x-ray
Normal	244	252
Carcinoma	38	36
Polyp	35	34
Crohn's disease	20	16
Ulcerative colitis	27	24
Diverticulitis	4	6
Total	368	368

Pathologies of colon detected by the hydrocolonic sonography are compared with the diagnosis established by double contrast barium enema x-ray.

### **V-3: GROUP-II PATIENTS:**

Total 140 patients were included into this group. After proper preparation all the patients were examined by hydrocolonic sonography. Proper hard copy recordings were obtained. After completing the examination, the patients were sent for double contrast barium enema x-ray of colon. X-ray films were obtained for hard copy documentation. Diagnoses were made based on the sonographic and radiographic findings of the examinations respectively. All the patients were then either examined by colonoscopy and proper biopsies were obtained. And or surgery was done and proper diagnosis was established by histopathological confirmation. HS and BEX diagnoses were correlated and compared with the diagnoses done by colonoscopy and or surgery. Here colonoscopy and or surgery diagnoses were regarded and gold standard. The results were presented in a tabulated for in Table – V-2.

Nosologic sensitivity, nosologic specificity, diagnostic sensitivity and diagnostic specificity of HS and BEX were calculated against the diagnosis done by colonoscopy and or surgery. Calculations were done according to the formula laid down in the Section IV-7 of Method chapter. Predictive value of negative HS & BEX and predictive value of positive HS & BEX were also calculated accordingly. All the results were presented in a tabulated for in the Table – V-3.

Table – V-2

	Colonoscopy & surgery FD	Hydrocolonic sonography		Double contrast barium enema x-ray	
		PFD [86]	NFD [54]	PFD [86]	NFD [54]
Carcinoma of colon	+ 29	+ 27	+ 3	+ 24	+ 6
	- 111	- 2	- 108	- 5	- 105
Polyp of colon	+ 36	+ 33	+ 8	+ 30	+ 10
	- 104	- 3	- 96	- 6	- 94
Crohn's disease	+ 12	+ 12	+ 4	+ 9	+ 5
	- 128	- 0	- 124	- 3	- 123
Ulcerative colitis	+ 9	+ 8	+ 4	+ 8	+ 4
	- 131	- 0	- 127	- 0	- 127
Normal colon	54	6	35	15	29
Total	140	140		140	

Group-II: Diagnosis of normal colon and colon disease by [a] colonoscopy and or surgery, by [b] hydrocolonic sonography and by [c] double contrast barium enema x-ray. Diagnosis by [a] considered final diagnosis. HS and BEX diagnosis are compared with [a]. PFD = Positive final diagnosis done by [a]. NFD = Negative final diagnosis done by [b]. + = Positive diagnosis, - = Negative diagnosis.

Table – V-3

	Nosologic sensitivity %		Nosologic specificity %		Diagnostic sensitivity %		Diagnostic specificity %	
	HS	BEX	HS	BEX	HS	BEX	HS	BEX
Carcinoma of colon	93.10	82.76	97.30	94.49	98.18	95.45	90.00	80.00
Polyp of colon	91.67	83.33	92.31	90.38	96.97	94.00	80.49	75.00
Crohn's disease	100.00	75.00	96.87	96.09	100.00	97.62	75.00	64.28
Ulcerative colitis	88.89	88.89	96.95	96.95	100.00	100.00	66.67	66.67

Group-II: Nosologic sensitivity, nosologic specificity, diagnostic sensitivity and diagnostic specificity of hydrocolonic sonography diagnoses [HS] and double contrast barium enema x-ray diagnoses [BEX] were calculated against the diagnoses done by colonoscopy and or surgery. Calculations are done according to the formula laid down in the Section IV-7 of Method chapter

---

**DISCUSSIONS**  
**CHAPTER 6**



## **VI: DISCUSSIONS**

### **VI-1: DISCUSSIONS OF THE FINDINGS:**

Prof. Bernd Limberg of Germany has developed the procedure of examining colon after instilling water into it [Limberg 1987]. Thereafter he has published a number of articles in English and in Germany. But the technique did not gain significant popularity. Few more authors have published their works in the subsequent years. But the work and publication of Prof. Bernd Limberg remained as major works in this field.

Instilling water into rectum as an adjunct to pelvic ultrasound was introduced as early as 1978 to detect origin of doubtful pelvic masses (Rubin et al 1978). Jasinski et al used a similar technique to demonstrate colon wall with little usefulness (Jasiniski et al 1981).

In our study we examined 523 patients by hydrocolonic sonography. Out of 523 patients, in 508 [97.13%] patients the entire colon starting at rectosigmoid junction and ending at the caecum could be visualized. Per abdominal examinations were started as soon as water began to instill. So, it was possible to monitor the passage of water through anus into colon. Water

was not instilled too hurriedly, as because this might produce some extra discomfort and sense of defaecation to the patient. More over whenever the patient felt discomfort we stopped water instillation temporarily and the examination started a few minutes later. Most of the time this procedure was enough to complete the examination. In 21 cases, the patient really went to toilet and the examination restarted after he coming back.

In the 15 [2.87%] of the patients the technique was partially useful mainly due to non-cooperation of the patients. The preparation was not adequate and the patient refused to take further preparations and the examination discontinued. The patient's preparation originally followed by Limberg and many other authors was lengthy, time consuming and difficult. We followed a different but simplified method. In fact this method of patient's preparation already existed in the radiological departments of our country for many years. This method of giving warm soap water enema was effective in most of the cases. 1 to 2 enemas were sufficient to prepare the patient satisfactorily. A few patients needed more than 2 enemas, but their number was not significant. More over presence of small amount of stool did not interfere with the examination. As because the real-time scanner could differentiate stool from polyp easily. Stool was movable and easily detachable from the wall, which was not true for the polyp.

No significant adverse reaction was present during or after the procedure. Few patients experienced mild to moderate discomfort. In only a few cases the examination was discontinued to be started after some time. The procedure was well tolerated and no hyasomide drug was given to the patients. The previous authors had given hyosomide injection routinely to all the patients prior to the examination. In our experience we saw that, most of the patients disliked more to have injection than that of the examination itself. Theoretically, there might have some possibility of absorbing excess water in colon. But the examination time was only 15 to 20 minutes and there was no chance of water intoxication.

In our study we wanted to set a criteria of normality and abnormality of colon by HCS. Normal colon wall found up to 4mm thick which conforms to previous report by Limberg and by the other authors doing hydrocolonic sonography (Limberg & Osswald 1994, Limberg 1992, Limberg 1990 a, Limberg 1990 b, Limberg 1989, Limberg 1988, Limberg 1987, Limberg 1999, Walter et al 1992, Elewaut et al 1995, Hernandez-Socorro et al 1995, Hiroki et al 1989, Bala et al 1999, Bala et al 2000, Bala et al 2001)..

All the five layers were clearly identified. The ileocaecal valve was visualized. Cases with polyp, carcinoma, ulcerative colitis and Crohn's disease were diagnosed by HCS and as good as double contrast barium

enema x-ray. In the Group-I patients, our experience was for detecting carcinoma, polyp, Crohn's disease and ulcerative colitis HS was equally, even more, effective than that of BEX. But for detecting diverticulitis, BEX was better than that of HC.

In the Group-II patients the findings were compared with a gold standard set by colonoscopy with biopsy and or surgery with histopathological confirmation. Total 140 patients were examined, 54 had normal colon and 86 had some type of colon pathology. We had tried to avoid ambiguity and made some clear-cut diagnosis. In many of the cases, the disease process was in advanced stage and patient went for surgery. In diagnosing carcinoma colon, polyp and Ulcerative colitis HS was better than that of BEX. But in case of ulcerative colitis it was equally effective.

The nosologic sensitivity and specificity of HS were between 88.89 to 100.00 and 92.31 to 97.30. Whereas of BEX it were between 75.00 to 88.89 and 90.38 to 96.09 respectively. The diagnostic sensitivity and specificity of HS were between 98.18 to 100.00 and 66.67 to 90.00 respectively. Whereas of BEX it were between 94.00 to 100.00 and 64.28 to 80.00 respectively. Predictive value of positive tests and predictive value of negative tests were also calculated and same as above results. The nosologic sensitivity and specificity, diagnostic sensitivity and specificity, predictive values of

positive and negative cases were in highly acceptable level, comparable and even better in comparison to BEX.

According to our study it is evident hydrocolonic sonography can be used instead of double contrast barium enema x-ray. More over colon wall total thickness and surrounding areas were covered by HS. In some cases, patients were referred for suspected mass in colon. But in HS it was seen that, colon was completely normal. Instead, in two cases liver masses at left lobe were detected. In another case suspected mass was in stomach antrum.

In an attempt to evaluate the ultrasound appearance *in vitro*, eight normal colon resection specimens, five specimens of colon carcinoma, two specimens of colon polyp one ulcerative colitis and one Crohn's disease were examined.

The controls were tumor-free resections of patients with colon tumors. The histological findings were compared with the sonographic findings. In addition, those patients with colon mass received a preoperative colon sonography examination. After a retrograde instillation the images of *in vitro* examinations were compared with *in vivo* images. The ultrasound findings of the colon specimens totally conformed to the findings found in *vivo* colon.

## **VI-2: CONFIRMATION WITH OTHER STUDIES:**

Prof. Bernd Limberg since his first article published in 1987 did extensive study on the topic. He published a lot of articles. All the articles conform to the result of our study. Hydrocolonic sonography is a valuable tool for examining colon in normal and in diseases. He had done ultrasonography of colon both in vivo and in vitro. Our in vitro study of colon specimen also gave the result with equal accuracy.

Prof. Limberg in his early work used difficult and time-consuming patient's preparation. This was done as because patients were prepared for colonoscopy and then examined with hydrocolonic sonography. But in subsequent years he gradually shifted to a more simple method of patient's preparation (Limberg 1987, Limberg 1988, Limberg 1994).

Prof. Limberg had developed expertise to examine and differentiate Crohn's disease and ulcerative colitis. In our study we did not get same level of sensitivity and specificity of examining Crohn's disease and ulcerative colitis. Most likely the prevalence of these diseases are less in our country than that of in Germany and we got less number of patient's with Crohn's disease and ulcerative colitis.

Hirooka et al did a study on hydrocolonic sonography (Hirooka et al 1989). But instead of instilling water through anus, he used oral water

administration. Magnesium citrate was used to enhance peristalsis and allowing water to come into colon before absorption. His results were in favor of our study.

Walter et al did a study in this subcontinent in Christian Medical College Hospital, Vellore. They also used lengthy preparation for the patients. They gave special emphasis on colon tuberculosis (Walter et al 1993). Prof. Limberg also requested us to give emphasis on colon tuberculosis. But we could not do that. As because findings of colon tuberculosis are ill defined and ambiguous until late stage. This part was kept for future study.

Carmen Rosa and co-workers tried to do staging of colon carcinoma by hydrocolonic sonography. They concluded, HS allows the diagnosis and preoperative staging of colorectal carcinomas with high sensitivity and specificity and predictive value. They recommended its preoperative use because of its definitive influence on surgical approach (Hernandez-Socorro et al 1995).

David Chi and co-workers, of course, got different results (Chui et al 1994). They compared their results with that of colonoscopy. But we compared usefulness of HS with that of BEX. Usefulness of colonoscopy for detecting colon pathology still remained as gold standard. Not only that, colonoscopy

can provide therapeutic options in addition to its diagnostic purpose. We found HS is as good as, even better, than that of BEX.

On the other hand, Elewaut and co-workers describe HS as a novel screening method for the detection of colon disease (Elewaut et al 1995). They had conducted an extensive study in Belgium and found HS very much useful for diagnosis of colon diseases.

Nosologic sensitivity and specificity, diagnostic sensitivity and specificity, predictive value of positive and negative results of our study is comparable with that of other study done by different workers of different places. Even in the 9<sup>th</sup> World Congress of AFSUMB in Florence, one paper on HS was presented by a doctor from Uganda. The work was done in Uganda and found very much suitable for their country.

HS is a promising technique of examining colon. But sufficient work was not done on it to establish its usefulness universally.



### **VI-3: LIMITATIONS:**

In Group-I patients we could not compare the results with some gold standard. Lack of adequate facilities, non-cooperation of the patients were contributing factors for this short comings. In Group-II patients we could compare all the results of HS and BEX with that of colonoscopic and surgical confirmations.

We could not include patients with significant illness into the study. We believe that, if there were hospital backups we could include almost all types of patients in the study. In our opinion, there was no absolute contraindication for hydrocolonic sonography.

Patients with colon tuberculosis could not be diagnosed. Because the findings were non-specific and ambiguous. Walter and co-workers has done some progress in this field. We kept this part for future research.

Follow up of the patients was a difficult task. This is an inherent difficulty in our country. There was no systematic way to trace patients for subsequent follow up. For this reason we have failed to include many patients in Group-II. Many times patients were reluctant to have further investigations and treatment.

#### **VI-4: RECOMMENDATIONS:**

A multi-centered study with large number of patients and facilities to establish confirm diagnosis is recommended. Other diagnostic facilities like CT and endoscopic sonography may be added in the study. Examination with probes of different frequencies should be done for every patient.

Power Doppler and Color Doppler may be a useful adjunct to the examination. Tissue harmonic scanning is now in use. This particular type of instruments may be proved more informative in hydrocolonic sonography.

Water is an excellent contrast agent. It is easily available and add no extra costs. But in some parts of the world echogenic oral contrast are tried for examining stomach and intestines. This echogenic contrast agent can be tried instead of water to evaluate which one is better.

Hydrocolonic sonography is a contrast enhanced ultrasonography. It is a new and developing branch of ultrasonography. Many things can be done. We recommend continuous updating of hydrocolonic sonography.

# CONCLUSIONS

---

## CHAPTER 7

## **VII: CONCLUSIONS**

### **VII-1: SUMMARY:**

The problem of the study was to determine if ultrasonography of colon could be done after instilling water into it and diagnosing colon diseases. Ordinarily colon can not be examined effectively by ultrasound. Instillation of water helps identifying and examining colon. Here water acts as an contrast agent.

The subjects of study were 523, of them 508 were examined fully. Rest 15 was dropped from the study due to inadequate preparation. Out of 508 patients, 457 were male and 51 were female.

The patients were referred from different Institutions in Dhaka area and from private settings. All the patients were examined in Institute of Nuclear Medicine and Training & Research Centre of the Bangladesh Institute of ultrasound in Medicine & Research. Subjects were divided into Group-I [368] and Group-II [140]. Group-I patients were examined by hydrocolonic

sonography and double contrast barium enema x-ray. Group-II patients were examined by HS, BEX with confirmation by colonoscopy and or surgery.

After a preparation, colon was examined by ultrasound after instilling water into colon through anus using a can and stand. Continuous examination was done as soon as the water was instilled.

BEX was done to every patient with the same preparation in the same day. If the examination was done in a different day, fresh preparation was given.

9 colon specimens were also examined by using water bath. In vivo findings were compared with in vitro findings.

The data were analysed by statistical techniques. Nosologic sensitivity, nosologic specificity, diagnostic sensitivity, diagnostic specificity, predictive value of positive results, predictive value of negative results were calculated.

## **VII-2: FINDINGS:**

1. All the patients could be examined from rectosigmoid to the caecum without difficulty.
2. Only an insignificant number of patients were dropped from the study due to inadequate preparation and the patient refused to take further preparations.

3. All the patients could tolerate the procedure quite well. No significant adverse effect noticed during and after the examination.
4. The findings were comparable with that of the double contrast barium enema x-ray. Even in some instances HS was better in comparison to BEX.
5. Colonoscopy and biopsy remained as the gold standard of diagnosis.

### **VII-3: CONCLUSIONS:**

1. Hydrocolonic sonography is a significant useful diagnostic imaging modality to evaluate colon diseases.
2. Diagnostic sensitivity and specificity of HS are comparable, even better, in comparison to double contrast barium enema x-ray.
3. The procedure is well tolerated and acceptable to the patients. No significant adverse effect is noticed during or after the examination.
4. There is no ionizing radiation for the ultrasound examination.
5. The modality is cheap and without any adverse effect.
6. The examination of colon by HS can be introduced in the country without any significant involvement of extra money or man-power.

#### **VII-4: IMPLEMENTATIONS:**

1. Hydrocolonic sonography should be introduced as a routine diagnostic imaging modality of colon diseases.
2. All the Government hospitals, Institutions, Private settings can provide the service in the out patient departments.

#### **VII-5: RECOMMENDATIONS FOR FURTHER STUDIES:**

1. A multi-centered study with large number of patients and facilities can be done.
2. CT and endoscopic sonography facilities may be incorporate.
3. Power Doppler and Color Doppler facilities should be incorporated with the future study.
4. Tissue harmonic scanner should be included in the study.
5. Different types of contrasts instead of water should be tried for better result.
6. Study should be done in hospital settings to include moderate to severe ill patients in the study.

REFERENCES  

---

CHAPTER 8



## VIII. REFERENCES

1. Alexander AA, Miller LS, Liu J-B, Feld RI, Goldberg BB. High resolution endoluminal sonography of the anal sphincter complex. *J Ultrasound Med* 1994a; 13:281-4.
2. Alexander AA, Liu J-B, Palazzo JP, et al. Endorectal color and duplex imaging of the normal rectal wall and rectal masses. *J Ultrasound Med* 1994b; 13: 509-15
3. Allan RN, Pease P, Ibbotson JP. Clustering of Crohn's disease in a Cotswold village. *Quarterly Journal of Medicine* 1986; 59: 473 – 8
4. Archampong EQ, Harris J, Clark CG. 1972 Absorption and secretion of water and electrolytes across the healthy and the diseased human colonic mucosa measured in-vitro. *Gut* 1972; 13: 880 – 6
5. Atkin WS, Morson BC, Cuzick J. Long-term risk of colorectal cancer after excision of rectosigmoid adenomas. *N Engl J Med* 1992; 26: 658
6. Audie JP, Janin A, Porchet N, Copin MC, Gosselin B, Aubert JP. Expression of human mucin genes in respiratory, digestive and reproductive tracts ascertained by in situ hybridisation. *Journal of Histochemistry and Cytochemistry* 1993; 41: 1479 – 85
7. Batty GM, Wilkins WE, Morris JS. Ulcerative colitis in a husband and wife. *Gut* 1994; 35: 562 – 3

8. Bergstrand O, Hellers G. Breast-feeding during infancy in patients who later develop Crohn's disease. *Scandinavian Journal of Gastroenterology* 1983; 18: 903 – 6
9. Bianchi PG, Panza E. Smoking, sugar and inflammatory bowel disease. *British Medical Journal* 1985; 291: 971 – 2
10. Bluth EI, Merritt CRB, Sullivan MA. Ultrasonic evaluation of the stomach, small bowel, and colon. *Radiology* 1979; 133: 677-80
11. Bolondi L, Caletti G, Casanova P, et al. Problems and variations in the interpretation of the ultrasound feature of the normal upper and lower gastrointestinal tract wall. *Scan J Gastroenterol* 1986; 21: 16-26
12. Bonfils S, Hervior P, Giroder J, Le Quintrec Y, Boucher JP, Gastard J. Acute spontaneously, recovering ulcerative colitis. *Digestive Diseases* 1977; 22: 429 – 36
13. Brandes JW, Stenner A, Martini GA. Ernährungsgewohnheitender Patienten mit Colitis ulcerosa. *Zeitschrift für Gastroenterologie* 1979; 17: 834 – 842.
14. Broberger O, Perlmann P. Autoantibodies in human ulcerative colitis. *Journal of Experimental Medicine* 1959; 110: 657 – 673.
15. Chess S, Chess D, Olander G, Benner W, Cole WH. Production of chronic enteritis and other systemic lesions by ingestion of finely divided foreign materials. *Surgery* 1950; 27: 221 – 34
16. Chui DW, Gooding GAW, McQuaid KR, Griswold V, Grendell JH. Hydrocolonic polyps and tumors. *N Engl J Med* 1994; 331: 1685-8
17. Clark, JC, Collan Y, Eide TJ, et al. Prevalence of polyps in an autopsy series from areas with varying incidence of large-bowel cancer. *Int. J. Cancer* 1985; 36: 179

18. Coombe C, Saunders W. A singular case of stricture and thickening of the ileum. *Medical Transactions of the Royal College of Physicians, London* 1813; 4: 16 – 8
19. Correa, P. Epidemiology of polyps and cancer. In Morson BC (ed.), *The Pathogenesis of Colorectal Cancer*, Philadelphia, W. B. Saunders 1978; 126
20. Crohn B B, Ginzburg L, Oppenheimer G D. Regional ileitis: a pathologic and clinical entity. *Journal of the American Medical Association* 1932; 106: 1.
21. Dalziel TK. Chronic interstitial enteritis. *British Medical Journal* 1913; ii: 1068 – 70
22. Darchis I, Colombel JF, Cortot A, Devred M, Paris JC. Crohn's disease in a married couple and their four children. (Letter). *Lancet* 1989; 737.
23. Das KM, Dubin R, Nagai T. Isolation and characterization of colonic tissue-bound antibodies from patients with idiopathic ulcerative colitis. *Proceedings of the National Academy of Sciences of the United States of America* 1978; 75: 4528 – 32
24. Deen KI, Madoff RD, Belmonte C, Wong WD. Preoperative staging of rectal neoplasms with endorectal ultrasonography. *Semin Colon Rectal Surg* 1995; 6: 78-85
25. Detry RJ, Kartheuser A, Cessions PJ. Endorectal ultrasonography for staging small rectal tumours: technique and contribution to treatment. *World J Surg* 1993; 17: 271-6
26. DiCandio G, Mosca F, Campatelli A, et al. Sonographic detection of postsurgical recurrence of Crohn's disease. *AJR* 1986; 146: 523-6
27. DiSario JA, Foutch PG, Mai HD. Prevalence and malignant potential of colorectal polyps in asymptomatic average-risk men. *Am. J. Gastroenterol.* 1991; 86: 941

28. Dubbins PA. Ultrasound demonstration of bowel wall thickness in inflammatory bowel disease. *Clin Radiol* 1984; 35: 227-31
29. Duthie HL, Watts JM, De Dombal FT, Golligher JC. Serum electrolytes and colonic transfer of water and electrolytes in chronic ulcerative colitis. *Gastroenterology* 1964; 47: 525 – 30
30. Edmons CJ, Pilcher D. Electrical potential differences and sodium and potassium fluxes across rectal mucosa in ulcerative colitis. *Gut* 1973; 14: 784 – 9
31. Eide T J, Stalsberg H. Polyps of the large intestine in northern Norway. *Cancer* 1978; 42: 2839
32. Ekbon A, Helmick C, Zack M, et al. Ulcerative colitis and colorectal cancer. A population based study. *N Engl J Med* 1990; 323: 1228
33. Elewaut AE, Afschrift M. Hydrocolonic sonography: A novel screening method for the detection of colonic disease? *Bildgebung* 1995; 62: 230-4
34. Fuchs CS, Giovannucci EL, Colditz, GA, et al. A prospective study of family history and the risk of colorectal cancer. *N Engl J Med* 1994; 331: 1969
35. Gambús G, De Bolos C, Andreu D, Franci C, Egea G, Real FX. Detection of the MUC2 apomucin tandem repeat with a mouse monoclonal antibody. *Gastroenterology* 1993; 104: 93 – 102.
36. Glassman MS, Newman LJ, Berezin S, Gryboski JD. Cow's milk protein sensitivity during infancy in patients with inflammatory bowel disease. *American Journal of Gastroenterology* 1990; 85: 838 – 40.

37. Granqvist, S. Distribution of polyps in the large bowel in relation to age: A colonoscopic study. *Scand. J. Gastroenterol.* 1981; 16: 1025.
38. Grisham MB. Role of reactive oxygen metabolites in inflammatory bowel disease. *Current Opinion in Gastroenterology* 1993; 9: 971 – 980.
39. Habershon SO. Diseases of the abdomen. *J Churchill*, London Harris FI, Bell GH, Brunn H. Chronic cicatrizing enteritis. *Surgery, Gynecology and Obstetrics* 1933; 57: 637 – 45
40. Harries AD, Baird A, Rhodes J. Non-smoking: a feature of ulcerative colitis. *British Medical Journal* 1982; 284: 706
41. Harris J, Shields R. Absorption and secretion of water and electrolytes by the intact human colon in diffuse untreated proctocolitis. *Gut* 1970; 11: 27 – 33
42. Hawker P C, McKay J S, Turnberg L A 1980 Electrolyte transport across colonic mucosa from patients with inflammatory bowel disease. *Gastroenterology* 79: 508 – 511.
43. Harris ML, Schiller HJ, Reily PM, Donowitz M, Grisham MB, Bulkley GB. Free Radicals and other reactive oxygen metabolites in inflammatory bowel disease. Cause, consequence, or epiphenomenon? *Pharmacology and Therapeutics* 1992; 53: 375 – 408.
44. Hawker PC, McKay JS, Turnberg LA. Electrolyte transport across colonic mucosa from patients with inflammatory bowel disease. *Gastroenterology* 1980; 79: 508 – 11
45. Heilbrun K, Nomura A, Hankin TH, et al. Diet and colorectal cancer with special reference to fiber intake. *Int J Cancer* 1989; 47: 1

46. Hernandez-Socorro CR, Guerra C, Herrandez-Romero J, et al. Colorectal carcinomas: Diagnosis and preoperative staging by hydrocolonic sonography. *Surgery* 1995; 117: 609-15
47. Heyder N, Kaarmann H, Giedl J. Experimental investigations into the possibility of differentiating early from invasive carcinoma of the stomach by means of ultrasound. *Endoscopy* 1987; 19: 228-32
48. Hildebrandt U, Feifel G, Schwarz HP, Scherr O. Endorectal ultrasound: instrumentation and clinical aspects. *Int J Colorectal Dis* 1986; 1: 203-7
49. Hirooka N, Ohno T, Misonoo M, et al. Sono-enterocolonosonography by oral water administration. *Clin Ultrasound* 1989; 17: 585-9
50. Ho SB, Chandler DJ, Logan G, Toribara NW, Kim YS, Ewing SL. Intestinal and gastric mucin core peptide expression in colon cancer: correlation with histology, stage and survival. *Gastroenterology* 1999; 104 (4), A 410
51. Hulsmans FH, Tio TL, Mathus-Vliegen EMH, Bosma A, Tytgat GNJ. Colorectal villous adenoma: transrectal US in screening for invasive malignancy. *Radiology* 1992; 185: 193-6
52. Hulsmans FJH, Mathus-Viegen LMH, Bosman S, Bosma A, Tytgat GNJ. Colorectal adenomas: inflammatory changes that simulate malignancy after laser coagulation-evaluation with transrectal US. *Radiology* 1993; 187: 367-71
53. Katzka, I., Brody, R. S., Morris, E., et al. Assessment of colorectal cancer risk in patients with ulcerative colitis: Experience from a private practice. *Gastroenterology* 85:22, 1983

54. Kasper H, Sommer H. Taste thresholds in patients with Crohn's disease. *Journal of Human Nutrition* 1980; 34: 455 – 6
55. Kilpatrick ZM, Silverman JF, Betancourt E, Farman J, Lawson JP. Vascular occlusion of the colon and oral contraceptives. *New England Journal of Medicine* 1968; 278: 438-40
56. Kimmey MB, Martin RW, Haggitt RC, et al. Histologic correlates of gastrointestinal ultrasound images. *Gastroenterology* 1989; 96: 433-41
57. Klebanoff SJ. 1992 Oxygen metabolites from phagocytes. I: Galli J I, Goldstein I M, Snyderman R (eds) *Inflammation: basic principles and clinical correlates*. Raven Press, New York 1992; 541 – 88
58. Lagercrantz R, Hammarstrom S, Perlmann P, Gustafsson BE. Immunological studies in ulcerative colitis. 3. Incidence of antibodies to colon-antigenin ulcerative colitis and other gastro-intestinal diseases. *Clinical and Experimental Immunology* 1966; 1: 263 – 276.
59. Lennard Heron HC, Khubchandani IT, Trimpi HD, Sheets JA, Stasik JJ. Evanescent colitis. *Diseases of the Colon and Rectum* 1981; 24: 555 – 61
60. Lennard – Jones JE, Cooper GW, Newell AC, Wilson CWE, Jones FA. Observations on idiopathic proctitis. *Gut* 1962; 3: 201 – 6
61. Lennard-Jones JE, Melville DM, Morson BC, et al. Precancer and cancer in extensive ulcerative colitis: Findings among 401 patients over 22 years. *Gut* 1990; 31: 800
62. Lev R. *Adenomatous Ployps of the Colon: Pathological and Clinical Features*. New York. Springer-Verlag, 1990

63. Limberg B. Diagnosis of inflammatory and neoplastic colonic disease by sonography. *J Clin Gastroenterol* 1987; 9: 5: 607-11
64. Limberg B. Diagnosis of inflammatory and neoplastic large bowel disease by conventional abdominal and colonic sonography. *Ultrasound Quarterly* 1988; 6: 2: 151-66
65. Limberg B. Diagnosis of acute ulcerative colitis and colonic Crohn's disease by colonic sonography. *J Clin Ultrasound* 1989; 17: 25-31
66. Limberg B. Diagnosis of large bowel tumors by colonic sonography. *Lancet* 1990; 335: 144-6
67. Limberg B. Sonographic features of colonic Crohn's disease comparison *in vivo* and *in vitro* studies. *J Clin Ultrasound* 1990; 18:161-6
68. Limberg B. Diagnosis and staging of colonic tumors by conventional abdominal sonography as compared with hydrocolonic sonography. *N Engl J Med* 1992; 327: 65-9
69. Limberg B. Osswald B. Diagnosis and differential diagnosis of ulcerative colitis and Crohn's disease by hydrocolonic sonography. *Am J Gastroenterol* 1994, 89: 7: 1051-7
70. Limberg B. Diagnosis of chronic inflammatory bowel diseases by ultrasonography. *Z Gastroenterol* 1999; 37: 495-508
71. Littrup JP. The development of a three-dimensional prostate model. *Prog Clin Biol Res* 1987; 237: 213-8



72. Lobo AJ, Foster PN, Sobala GM, Axon ATR. Crohn's disease in married couples. *Lancet* 1988; 1: 704 – 5
73. Lutz H, Petzoldt R. Ultrasonic patterns of space occupying lesions of the stomach and the intestine. *Ultrasound Med Biol* 1976; 2: 129-31
74. Marcussen H. Anti-colon antibodies in ulcerative colitis. A clinical study. *Scandinavian Journal of Gastroenterology* 1976; 11: 763 – 767.
75. Martini GA, Brandes JW. 1976 Increased consumption of refined carbohydrates in patients with Crohn's disease. *Klinische Wochenschrift* 1976; 54: 367 – 71
76. Mayberry JF, Hitchens RAN. Distribution of Crohn's disease in Cardiff. *Social Science and Medicine (Oxford)* 1978; 12: 137 – 8
77. Miller LS, Liu JB, Colizzo FP, et al. Correlation of high frequency esophageal ultrasonography and manometry in the study of esophageal motility. *Gastroenterology* 1995; 109: 832-7
78. Montgomery RD, Frazer AC, Hood C, Goodhart JM, Holland MR, Schneider R. Studies of intestinal fermentation in ulcerative colitis. *Gut* 1968; 9: 521 – 6
79. Monsen U, Brostrom O, Nordenvall B, et al. Prevalence of inflammatory bowel disease among relatives of patients with ulcerative colitis. *Scandinavian Journal of Gastroenterology* 1987; 22: 214-8
80. Morgagni J B. *De Sedibu et causis morborum*. Remondini, Veice 1769
81. Muto T, Bussey HJR, Morson BC. The evolution of cancer of the colon and rectum. *Cancer* 1975; 36:2251

82. Neugut AI, Jacobson JS, Ahsan H, et al. Incidence and recurrence rates of colorectal adenomas: A prospective study. *Gastroenterology* 1995; 108: 402
83. Nugent FW, Haggit RC, Gilpin PA. Cancer surveillance in ulcerative colitis. *Gastroenterology* 1991; 100: 1241
84. Orholm M, Munkholm P, Langholz E, et al. Familial occurrence of inflammatory bowel disease. *New England J Med* 1991; 324: 84-8
85. Orrom WJ, Wong WD, Rothenberger DA, Jensen LL, Goldberg SM. Endorectal ultrasound in the preoperative staging of rectal tumors. *Dis Colon Rectum* 1990; 33: 654-9
86. Provenzale D, Kowdley KU, Arora S, et al. Prophylactic colectomy or surveillance for chronic ulcerative colitis? A decision analysis. *Gastroenterology* 1995; 109: 1188
87. Rankin RN, Fenster A, Downey DB, Munk PL, Levin MF, Vellett AD. Three-dimensional sonographic reconstruction techniques and diagnostic applications. *AJR* 1993; 161: 695-702
88. Rao SSC, Read NW, Holdsworth CD. Is the diarrhea in ulcerative colitis related to a failure of colonic salvage of carbohydrate? *Gut* 1987; 28: 1090 - 4
89. Rask-Madsen J, Hammersgaard EA, Knudsen E. 1973 Rectal electrolyte transport and mucosal permeability in ulcerative colitis and Crohn's disease. *Journal of Laboratory and Clinical Medicine* 1973; 81: 242 - 53
90. Read NW. 1982 Diarrhoea. The failure of colonic salvage. *Lancet* 1982; ii: 481 - 3

91. Read NW, Johnson AJ. Disturbance of intestinal motor activity. In: Allan RN, Keighley MRB, Alexander-Williams J, Hawkins C (eds) *Inflammatory bowel disease*. Churchill Livingstone, Edinburgh 1983; 54 – 62
92. Reed JF III, Calkins BM, Rosen L. Concordance of familial characteristics in Crohn's disease and ulcerative colitis. *Disease of the Colon and Rectum* 1992; 35: 405 – 10
93. Reddy BS, Sharma C, Simi B, et al. Metabolic epidemiology of colon cancer: Effect of dietary fiber on fecal mutagens and bile acids in healthy subjects. *Cancer Res* 1987; 47: 644
94. Reilly RP, Robinson TJ. Crohn's disease- is there a long latent period? *Postgraduate Medical Journal* 1986; 62: 353 – 4
95. Reilly RP, Robinson TJ. 1986 Crohn's disease- is there a long latent4.
96. Rosch T, Classon M. *Gastroenterologic Endosonography*. Thieme Medical Publishers Inc.: New York 1992
97. Rustgi AK. Hereditary gastrointestinal polyposis and nonpolyposis syndromes. *N. Engl. J Med* 1994; 331
98. Samuelsson SM. 1976 Ulcrative colitis in the country of Uppsala 1945 0 1964. MD Thesis, Uppsala. Somerville KW, Logan RFA, Edmond M, Langman MJS. Smoking and Crohn's disease. *British Medical Journal* 1976; 289: 954 – 6
99. Sarrazin J, Wilson SR. Manifestations of Crohn Disease at US. *RadioGraphics* 1996; 16: 499-520.
100. Satsangi J, Jewell D P, Rosenberg W M C, Bell J I. Genetics of inflammatory bowel disease. *Gut* 1994; 35: 696 – 700

101. Seitz K, Rettenmaier G. Inflammatory bowel disease Sonographic Diagnostics. Dr. Falk Pharma. West Germany GmbH, 1988
102. Shike M, Winawer SJ, Greenwald PH. Primary prevention of colorectal cancer. Bull. WHO 1990; 68: 377
103. Shimizu S, Tada M, Kawai K. The value of endoscopic ultrasonography in the assessment of inflammatory bowel disease. Endoscopy 1992; 24 (suppl 1): 254-8
104. Snook JA, de Silva HJ, Jewell DP. The association of autoimmune disorders with inflammatory bowel disease. Quarterly Journal of Medicine 1991; 72: 835 – 840.
105. Snook JA, Lowes JR, Wu KC, Priddle JD, Jewell DP 1991 Serum and tissue autoantibodies to colonic epithelium in ulcerative colitis. Gut 1991; 32: 163 – 6
106. Stemmermann GN, Yatani R. Diverticulosis and polyps of the large intestine: A necropsy study of Hawaii Japanese. Cancer 1973; 31: 1260
107. St. John D JB, McDermott FT, Hpper J L, et al. Cancer risk in relatives of patients with common colorectal cancer. Ann Intern Med 11993; 18: 785
108. Sullivan SN. Hypothesis revisited: toothpaste and the cause of Crohn's disease. Lancet 1990; 336: 1096 – 7
109. Tedesco FJ, Valpolicelli NA, Moore FS. Oestrogen and progesterone associated colitis: a disorder with clinical and endoscopic features mimicking Crohn's disease. Gastrointestinal Endoscopy 1982; 26: 247 – 9
110. Thornton JR, Emmett PM, Heaton KW. Diet and Crohn's disease: characteristics of the pre-illness diet. British Journal 1979; 2: 762 – 4

111. Tysk C, Lindberg E, Jarnerot G, Floderus-Myrhed B. Ulcerative colitis and Crohn's disease in an unselected population of monozygotic and dizygotic twins. A study of heritability and the influence of smoking. *Gut* 1988; 29: 990 – 6
112. Vatn MH, Stalsberg H. The prevalence of polyps of the large intestine in Oslo: An autopsy study. *Cancer* 1982; 49: 819
113. Walter DF, Govil s, William RR, Bhargava N, Chandy G. Colonic sonography: Preliminary observations. *Clin Radiology* 1992; 47: 200-4
114. Wang KY, Kimmery MB, Nyberg DA, et al. Colorectal neoplasms: accuracy of US in demonstrating the depth of invasion. *Radiology* 1987;165: 827-9
115. Whorwell PJ, Holdstock G, Whorwell GM, Wright R. Bottle feeding, early gastroenteritis, and inflammatory bowel disease. *British Medical Journal* 1979; 1: 382.
116. Wilks S, Moxon W. Lectures on pathological anatomy, 2nd edn. Lindsay and Blakiston, Philadelphia 1875, pp 408 -- 9
117. Itzkowitz, SH. Gastrointestinal adenomatous polyps. *Semin. Gastrointest. Dis.* 1996; 7:105
118. Wilson SR. Gastrointestinal sonography. *Abdom Imaging* 1996; 21: 1-8
119. Winamar S., Zauber AG, Cercles H, et al. Risk of colorectal cancer in families of patients with adenomatous polyps. *N Engl J Med* 1996; 334: 82
120. Worliceck H, Lutz H, Heyder N, et al. Ultrasound findings in Crohn's disease and ulcerative colitis: A prospective study. *J Clin Ultrasound* 1987; 15: 153-63

121. Ziegler RG, De Vesa SS, Fraumeni J. Epidemiologic patterns of colorectal cancer. In Devita VT, Hellman S, Rosenberg SA, (eds.), *Important Advances in Oncology*. Philadelphia, JB Lippincott 1986; 209

---

**APPENDIXES**  
**CHAPTER 9**

**IX: APPENDICES – A****HYDROCOLONOSONOGRAPHY  
HISTORY SHEET & CONSENT OF PATIENT****ULTRASONOGRAPHY OF COLON**

Registration No.:

Date:

রোগীর নাম Name of the Patient	
বয়স ও লিঙ্গ Age and Sex	
পেশা Occupation	
বাসস্থান Residence	
ডাক্তারের নাম Doctor's Name	
প্রতিষ্ঠানের নাম Institution's Name	
প্রধান অসুবিধা Chief Complains	*
	*
	*
পাতলা /ফর্ম পায়খানা Diarrhea/ Constipation	
ব্যথা Pain	
রক্ত / আমপড়া Blood/Mucus	
পেটে চাকা / পানি Mass/Ascites	
পরীক্ষার ফলাফল Investigations	*
	*
	*
রোগ নির্ণয় Provisional Diagnosis	

আমি সম্পূর্ণ বুঝিয়া, নিজ ইচ্ছায়, পায়ুপথে নল দ্বারা পানি প্রবেশ করাইয়া বৃহৎ এর আল্ট্রাসোনোগ্রাফী  
পঞ্জীকর করাইতে সম্মত আছি।

I have understood the whole procedure of the ultrasound examination of colon. Yes, I am willing to do  
ultrasound examination of colon by introducing water in colon through anus.

সাক্ষীর স্বাক্ষর  
Signature of the Witness

রোগীর স্বাক্ষর ও তারিখ  
Signature of the Patient with Date



## IX: APPENDIXES – B

### HYDROCOLONOSONOGRAPHY FINDINGS & IMPRESSIONS

#### ULTRASONOGRAPHY OF COLON

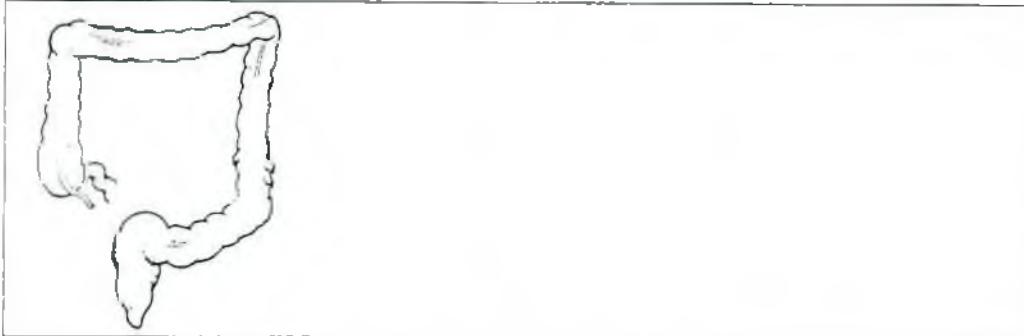
**FINDINGS:**

SIGMOID COLON		DESCENDING COLON	
Lumen Width		Lumen Width	
Wall Thickness		Wall Thickness	
Haustration		Haustration	
Wall Layers		Wall Layers	
Wall Pathology		Wall Pathology	
Outside Wall		Outside Wall	

TRANSVERSE COLON		ASCENDING COLON	
Lumen Width		Lumen Width	
Wall Thickness		Wall Thickness	
Haustration		Haustration	
Wall Layers		Wall Layers	
Wall Pathology		Wall Pathology	
Outside Wall		Outside Wall	

CAECUM		OTHERS	
Lumen Width			
Wall Thickness			
Haustration			
Wall Layers			
Wall Pathology			
Outside Wall			
Ileocaecal V.			

**COMMENT:**



**IX: APPENDIXES – C**

**HYDROCOLONOSONOGRAPHY  
REPORT PAGE-1**

**THE TRAINING & RESEARCH CENTRE**

**The Bangladesh Institute of Ultrasound in Medicine & Research**

[A Constituent Institute of the University of Science & Technology Chittagong]

7/8 Eastern Plaza, 6<sup>th</sup> Floor, Hatirpool, Dhaka 1205

Phone: 9668739, Fax: 880-2-8618297, E-mail: [bala@dhaka.agni.com](mailto:bala@dhaka.agni.com)

Patient's Name		Age:	Sex:
Referred By	Prof./Dr.		

No. ULTRASONOGRAPHY OF COLON Date:

DIAGNOSIS	

PROCEDURE :	A colonic preparation eliminates fecal materials and gases. By instilling water inside colon through anus, the whole length of colon is examined with a real-time ultrasound system using 5.0 MHz high-resolution curvilinear transducer.
-------------	---

SIGMOID COLON	Lumen Width	
	Wall Thickness	
	Haustration	
	Wall Layers	
	Wall Pathology	
	Outside Wall	

DESCENDING COLON	Lumen Width	
	Wall Thickness	
	Haustration	
	Wall Layers	
	Wall Pathology	
	Outside Wall	

**IX: APPENDIXES – D**

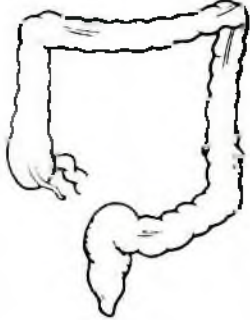
**HYDROCOLONOSONOGRAPHY  
REPORT PAGE-2**

TRANSVERSE COLON	Lumen Width	
	Wall Thickness	
	Haustration	
	Wall Layers	
	Wall Pathology	
	Outside Wall	

ASCENDING COLON	Lumen Width	
	Wall Thickness	
	Haustration	
	Wall Layers	
	Wall Pathology	
	Outside Wall	

CAECUM	Lumen Width	
	Wall Thickness	
	Haustration	
	Wall Layers	
	Wall Pathology	
	Outside Wall	
	Ileo-caecal Valve	

OTHERS		

LINE DRAWINGS	
---------------	--

Dr. Kanu Bala MBBS, DMUD

## IX: APPENDIXES – E

**Prof. Bernd Limberg, University of Göttingen, Germany  
Letter No. 1**

### **STÄDTISCHES KRANKENHAUS WOLFENBÜTTEL**

AKADEMISCHES LEHRKRANKENHAUS DER GEORG-AUGUST-UNIVERSITÄT  
GÖTTINGEN



Städtisches Krankenhaus | Postfach 1603 | 38299 Wolfenbüttel

Dr. Kanua G. Bala  
Institute of Ultrasound in Medicine  
University of science & Technology Chittagong  
10/26 Eastern Plaza, 9<sup>th</sup> Floor, Hatirpool  
Dhaka - 1205  
Bangladesh

Alter Weg 80  
D - 38302 Wolfenbüttel

Fachbereich  
**Medizinische Klinik**  
Chefarzt Prof. Dr. Limberg  
Auskunft erteilt:

Telefon: (05331) 934 - 0  
Durchwahl (05331) 934 - 361  
Fax: (05331) 934 - 369

Datum und Zeichen Ihres Schreibens

Mein Zeichen

Datum:  
1. 02. 01

Dear Doctor Bala!

Thank you for your letter and your reprints. I was glad to hear that you want to introduce hydrocolonic sonography in your country. It would be of special interest, whether hydrocolonic sonography is able to sonographically display tropical disease of the large bowel, i. e. intestinal tuberculosis and helminthism. I am very interested in your research and I would be glad to get the reprints of your papers.

Enclosed please find some reprints of my papers.

Sincerely yours

  
Prof. Dr. med. B. Limberg

## IX: APPENDIXES – F

**Prof. Bernd Limberg, University of Göttingen, Germany  
Letter No. 2**

### **STÄDTISCHES KRANKENHAUS WOLFENBÜTTEL**

AKADEMISCHES LEHRKRANKENHAUS DER GEORG-AUGUST-UNIVERSITÄT  
GÖTTINGEN

Städtisches Krankenhaus | Postfach 1863 | 38299 Wolfenbüttel

Dr. Kanu G. Bala  
Bangladesh Institute of Ultrasound  
10/26 Eastern Plaza, 9<sup>th</sup> Floor  
Hatirpool, Dhaka – 1206, Bangladesh



Alter Weg 50  
D - 38302 Wolfenbüttel

Fachbereich  
**Medizinische Klinik**  
Chefarzt Prof. Dr. Limberg  
Auskunft erteilt:

Telefon: (05331) 934 - 0  
Durchwahl (05331) 934 - 361  
Fax: (05331) 934 - 369

Daten und Zeichen Ihres Schreibens

Main Zeichen

Datum  
03 05 01

Dear Doctor Bala,

Thank you for your letter and the reprint of your interesting publication. The results of your study confirm the results of the study I published some years ago. These results demonstrate very clearly that hydrocolonic sonography is an alternative to the radiological examination of the colon. Furthermore, it is of great importance that hydrocolonic sonography is proven suitable for the examination of the colon in a developing country such as Bangladesh.

There is another study from India, which confirms your results as well (Dixit R, Chowdhury V, Kumar N.; Hydrocolonic sonography in the evaluation of colonic lesions. Abdom Imaging 1999; 24: 497 – 505).

Therefore, I strongly suggest that you should submit your paper for publication in „The Lancet“. From my point of view there is a good chance that your paper will be accepted. I am very interested to keep informed about the progress of your work.

Sincerely yours,

Prof. Dr. med. B. Limberg