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Video Capsule Endoscopy

A Reference Guide and Atlas

DVD-ROM
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Springer

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Foreword

Capsule endoscopy has been the most important innovation in endoscopy since video endoscopy replaced fiber optics. It has opened the whole of the small intestine to easy, direct, and safe endoscopic vision and in so doing has stimulated the development and increased the value of device-assisted enteroscopy. This has enabled minimally invasive intraluminal therapy to be performed throughout the whole of the gastrointestinal tract. The ability to visualize the small-bowel mucosa easily has also led to a better understanding of the pathological conditions that afflict the organ, has revolutionized the management of occult gastrointestinal hemorrhage, and has greatly improved the physician's ability to identify conditions such as Crohn's disease at an earlier stage and enables us to monitor its activity in response to treatment.

The development of enteroscopy however has highlighted some of the deficiencies inherent in endoscopic techniques and in particular the inability of optical visualization to provide information about disease within or outside of the bowel wall or, in the case of the small intestine, to determine exactly where in the intestine the pathology lies. The improvement of other imaging modalities, such as computed tomography and magnetic resonance, however has filled this gap, and our ability to link mucosal information to wider anatomical boundaries has further enhanced the contribution of enteroscopy to the study of the small intestine.

This remarkable book is more than an atlas of video capsule endoscopy. Its 52 chapters prepared by recognized experts comprehensively address all issues relating to capsule endoscopy, starting with the fascinating story of its development through the indications for its use, current equipment available, techniques, and training. A valuable chapter on the evaluation of capsule images brings the reader down to earth. Reading a huge number of images taken over the long period of the procedure requires software assistance, but even this cannot disguise the fact that the examination of images is time consuming for the reporting professional. Unlike real-time "hands-on" video endoscopy, it is not possible to view a lesion at leisure from different angles and distances or to touch it with biopsy forceps. Furthermore, the variety of different pathologies in the small bowel appears to be more extensive than those seen in the stomach or colon. Lesions are more difficult to identify than with video endoscopy as some of them are masked by the overlying villous mucosa. The images obtained are both fascinating and beautiful but not as crisp as those we have become used to with video endoscopy. Diagnosis is not a simple matter.

The second main section of the book addresses other modalities used to assess the small bowel. It includes excellent chapters on device-assisted enteroscopy as well as duodenoscopy and ileoscopy that detail the methodology and potential therapeutic applications again beautifully illustrated. Two chapters focus upon the use of computed tomography magnetic resonance and ultrasound, placing their use into the context of the management of small-bowel disease. The radiographs and scans, well annotated and labeled, enable the non-radiologist endoscopist reader to appreciate the abnormal findings with ease.

The central section of the book is devoted to the appearances of small-bowel lesions in a variety of conditions that include infections, systemic illness, and specific small-bowel conditions such as Crohn's, familial polyposis, rare lesions, and malignant tumors. The chapters comprise a cornucopia of high-quality common and uncommon small-bowel lesions accompanied by well-written explanation and background.

The next section of the book considers a variety of important areas, such as the prevention and management of complications and the use of capsule endoscopy in children. There is a particularly relevant and thoughtfully written chapter on the impact of capsule endoscopy on clinical outcome that puts the technique in context with other approaches to management and assesses its value in a variety of clinical conditions.

The last section first addresses the developing areas of esophageal capsule endoscopy and capsule endoscopy in the colon. It then moves on with two chapters that give a critical analysis of the deficiencies of the technology, among them considering the way that images might be improved and how the capsule could be independently controlled in real time. One chapter is on hardware, the other software.

This is altogether a most impressive book and one that should be available in all units that provide a capsule endoscopy service. It is of value both for reference purposes and as a learning experience for those starting in this area. It is particularly relevant for those in charge of endoscopy services if they are considering setting up a capsule endoscopy service. When capsule endoscopy was introduced, most endoscopists thought of it as a simple adjunct to conventional endoscopy and it would just be a matter of reviewing a number of images and making a diagnosis. This book pulls no punches. Capsule endoscopy is of great value, but those who are responsible for it require extensive training and experience. It is not for the dilettante.

The editors and authors are to be congratulated for this particularly important contribution to endoscopic literature.

Leeds, UK Anthony Axon, MD, FRCP

Preface

Since the publication of the *Atlas of Video Capsule Endoscopy* (M. Keuchel, F. Hagenmüller, and D. Fleischer) in 2006, video capsule endoscopy has steadily and rapidly developed. Technical improvements, extended scientific evidence, and availability of new capsule systems including colon capsules have required this update. The present book follows its predecessor by combining state-of-the-art presentation of the different aspects of capsule endoscopy with a comprehensive collection of typical and rare capsule images. Once again, a board of world-renowned experts have agreed to contribute their experience, images, and enthusiasm, which is very much appreciated.

A major goal of this atlas is, again, to help readers interpret a capsule endoscopy examination by relating the images to corresponding histologic, endoscopic, radiologic, surgical, and clinical reference findings. We thank all colleagues who generously provided many rare images and videos.

The support by our teams at Bethesda Hospital Bergedorf, Asklepios Klinik Altona, and Jikei University School of Medicine is gratefully appreciated.

We also thank Lee Klein of Springer in Philadelphia for his excellent coordination throughout the creation of this atlas, Bernice Wissler for outstanding copyediting, Dr. R. Nithyatharani of spi-global in Chennai for perfect layout and production, Patrick Waltemate of le-tex in Leipzig for realizing all ideas related to the DVD and Dr. Ute Heilmann and Martina Himberger of Springer in Heidelberg for their guidance through the production of the work. Last but not least, we thank our families for their support and tolerance as we prepared this book.

Hamburg, Germany — Martin Keuchel
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From Finding to Diagnosis

Synopsis of relevant findings in the small intestine: direct track to the image and video
See also:

Findings in the	
Normal small bowel	Chap. 19
Esophagus	Chaps. 42 and 43
Upper GI tract	Chap. 44
Colon	Chaps. 45 and 49
Postoperative GI tract	Chap. 39
Systematic description of findings	Chap. 10

Finding		Diagnosis	Image	Video
Stenosis	Extrinsic	Impression, bulge	10.2a	V19-20
	Duodenum	Peptic stenosis		V20-03
	Ulcerated	Crohn's	09.6	
		NSAIDs	30.9	
	Circular	NSAIDs	30.7	V16-02
	Edematous	Crohn's	25.10	V24.02
	Mucosal hemorrhage	Crohn's	25.9b	V25-01
	Petechiae	Crohn's	2.2	V24.03
		Ischemic enteropathy		V21-10
		Anastomotic stricture	39.13	
	Fibrotic	NSAIDs	30.8	V40-08
	White villi	Radiation enteritis	30.10	V30.06
	Erythematous, edema	Eosinophilic enteritis	27.8	
	Web like	NSAIDs	10.2b	V30-04
		Acute GI-GvHD	31.6	V31-01
		Follicular lymphoma	34.16	V34-11
		Anastomotic ulcer	39.14	V39-09
		EATL	34.19	
	Ulcerated infiltration	EATL	26.7	V34-12
		Centroblastic B-cell lymphoma	34.17	V40-07
	Mass	Adenocarcinoma	10.2c	V34-01
		Neuroendocrine carcinoma	34.6	V34-05
		Lymphoma	34.20	
		Metastasis	34.25-26	V34-16/17
Second lumen	Anastomosis	Roux-en-Y	10.3a	V39-03
	Circular ulcer	Side to side	39.14	V39-09
	Diverticulum	Duodenal	20.2	V20-01
		Jejunal	20.3	V20-02
		Meckel's	20.9	V20-05
		Anastomotic diverticulum	39.11	V39-06
		Side anastomosis	39.14	V39-09
		Duplication cyst	20.14	

	Fistula	Crohn's	49.8	V49-03 V25-02
	Artifact	Air bubble	05.19d	V05-17
Dilated lumen		Adhesion	39.22	V40-08
		Duodenojejunostomy	–	V39-02
		Roux-en-Y anastomosis	39.9	V39-03
Foreign body	Worm	Strongyloides	28.19	V28-06
	Tiny			
	Multiple, small, in colon	Pinworm	28.20	V28-07
	Multiple	Hookworm	28.23	
	Multiple, one thin end	Whipworm	28.21	V28-08
	Long	Roundworm	28.17	
	Long, multiple segments	Tapeworm	28.24-25	V28-09/10
	Capsules/remnants	Patency capsule	09.9	V09-01
		Patency capsule coating	09.10	V09-02
		PillCam	10.5b	V05-05
	Postinterventional	Intestinal suture	39.20c, d	
		Gastric suture	44.20	
		Staple	10.3b	V39-07
		Endoscopic clip	10.5a	V39.12
Intussusception	Idiopathic	Adolescent	38.15	V38-07
	Secondary	Ectopic pancreas	33.14-15	
		Lipoma	33.29	
		Hamartomatous polyp		V33-02
Edema	Diffuse	Portal hypertension	10.6b	V23-02
		Small-bowel transplantation	39.27	
	Glassy	Mycobacterium avium	28.2	V28-02
		Tuberculosis	28.3	
		Radiation enteritis	22.11	
	Erythema	Eosinophilic enteritis	27.8	
		IgA vasculitis	29.6c	V29-02
	Diffuse white villi	Whipple's disease	28.1	V28-01
		Yellow nail syndrome	22.15	
		Hypobetalipoproteinemia	29.18	
		Waldman's disease	38.14	
	Red spots	Systemic lupus erythematosus	29.13	
	Ulceration	Acute GI-GvHD	31.7	
	Bleeding	Acute ischemia	21.14	
	Segmental	Ileostoma	39.25a	
Abnormal mucosa	White, edematous	Primary lymphangiectasia	22.14	V22-02
		Hypobetalipoproteinemia	29.18	
		Yellow nail syndrome	22.15	
	Atrophic	Diverticulum	10.6d	
	Erosive	Acute GI-GvHD	31.3	
	Focally denuded	Crohn's	25.11	
		NSAIDs		V30-01/02
	Scalloped	Celiac	26.4	V26-03
	Reticulate	Portal hypertension	23.3-4	
	Nodular, ulcerated	Crohn's (cobblestone)	25.8b	V25-01
		Acute GI-GvHD	31.3-4	V31-01
		EATL	2.7	
	Granular	Autoimmune enteropathy	26.9	V26-10
	Hemorrhagic	Crohn's	25.9	
		Eosinophilic enteritis	27.9d	V27.01
		IgA vasculitis	29.5d	V29-02
	Ulcerated	IgA vasculitis	29.5f	V29-02
		Acute GI-GvHD	31.8	V31-01

	Friable	Eosinophilic enteritis	27.5	
		Noonan syndrome	29.22	
	Erythematous	Eosinophilic enteritis	27.5	
		NSAIDs	10.6c	V30-03
	Fibrotic	Panniculitis	28.10	V28-05
		Radiation enteritis	30.10	V30-05
	Atypical vessels	Systemic sclerosis	29.14d	
Atrophic villi	Patchy villous atrophy	NSAIDs	10.8	V30-01
	Patchy erythema	Crohn's	25.11	
	Patchy white villi	Crohn's	24.10	
		NSAIDs	30.1	
	Patchy erythema	Eosinophilic enteritis	27.3	
		NSAIDs		V30-03
	Erythema, edema, ulcer	Idiopathic chronic non-granulomatous jejunitis	50.11a	V26-11
		IgA vasculitis	29.5	V29-02
	Erosion	NSAIDs	30.2	
	Stenosis	NSAIDs	2.3b	
	Diffuse partial villous atrophy	Celiac	26.3b	
		Chemotherapy induced	30.12	
		Collagenous sprue	26.11	
	Nodules	CVID	29.16	
	Ulcerations	Ulcerative jejunoileitis	26.6	V26-08
	Diffuse total villous atrophy	Celiac sprue	26.3a	V26-01
	Fissures	Celiac sprue	02.6	V26-03
	White villi	Refractory sprue type II	26.5	
	Ulcerations	Refractory sprue type II		V26-06
		Autoimmune enteropathy		V26-09
	Ulceration, nodularity	EATL	02.7	
	Ulceration, stricture	EATL	34.19	
	Mass	EATL	34.20	
	Bleeding	Autoimmune enteropathy		V26-10
White villi	Diffuse white villi	Waldman's disease	22.14	V22-02
		Whipple's disease	28.1	V28-01
		Yellow nail syndrome	22.15	
		HIV	28.9	
	Nodular	Hennekam syndrome	22.16	V38-05
		Cronkhite-Canada syndrome	37.6	V37-04
	Granular	Sea blue histiocytosis	29.20	
	Patchy white villi	Atypical mycobacteriosis	22.6	V28-02
		Salmonellosis	22.7	
	Bleeding	Goldenhar syndrome	29.23	
	Nodules	Blastomycosis	28.14	
	Edema	Functional lymphangiectasia	22.1	
		Mycobacterium avium	28.2	V28-02
		Tuberculosis	28.3	
	Patchy villous atrophy	NSAIDs	30.1	V30-01
	Ulcer	NSAIDs	30.2	
	Ulcers, nodules	Idiopathic chronic non-granulomatous jejunitis	50.11a	V26-11
	Diffuse villous atrophy	Autoimmune enteropathy	26.9	V26-10
	Ulcers	Ulcerative jejunoileitis	26.6	V26-07
		Ischemic enteritis	21.15	V21-08
	Mucosal hemorrhage	Refractory sprue type II	26.5	
	Fibrosis	Radiation enteritis	30.10	V30-06
			22.12	

	Bleeding	Radiation enteritis	22.11	V30-05
	Aphthae, erosion	Crohn's disease	22.4	V24-01
		Collagenosis	22.5	
	Peritumorous	Adenocarcinoma	22.9	V34-01
		Lymphoma	22.8	V34-11
		Metastasis	22.10	V34-17
		Neuroendocrine carcinoma	34.6	V34-05
		GIST		V34-08
		MALT lymphoma	34.18	
White spots	Circumscript	Flat adenoma, sporadic	33.18	V33-03
Plaques		Flat adenoma (FAP)	35.6	V35-01
	Cystic	Lymph cysts	19.16	V19-16
	Solid, small	Lymph follicle	19.18a	V19-17
	Focal white villi	Focal lymphangiectasia	22.2	
	Plaque, irregular	Early duodenal carcinoma	34.1	
	Aphthae	Crohn's	25.6	V24-01
		Collagenosis	29.12	
	Patchy			
	Patchy white villi	Patchy lymphangiectasia	22.3	V22-01
	Patchy plaques	Acute GI-GvHD	31.9	V31-01
	Aphthae and nodules	Intestinal spirochetosis	28.6	V28-03
	Granula	Eosinophilic enteritis	27.9e	V27-01
	Segmental plaque, circular	Circular duodenal adenoma	33.19	V33-05
Black spot	Flat, large	Ink mark	39.31	V39-11
	Elevated	Melanoma	34.22	V34-13
	Multiple, diffuse	Hemosiderosis	29.17	V29-03
Red spots	Sharply demarcated	Angiectasia	10.9	V21-01 – 03
		In portal hypertension	23.5	
	Fern-like arborization	Angiectasia in HHT	21.2	V21-05
	With bleeding	Angiectasia, bleeding	21.3	
	Multiple, localized	Neuroendocrine carcinoma	5.23c	V34-06
	Unsharp	Atypical angiectasia	5.8cd	V05-06
		Crohn's	25.9a	
		Goldenhar syndrome	29.23	
		Refractory sprue type II	26.5	
	Erosion	NSAIDs	30.3	V30-02
	Ulcers	Amyloidosis	29.1	V29-01
		Cytomegalovirus	28.8	V28-04
		Crohn's	2.2	V24-03
	Erythema	Portal hypertension	23.4	
		Eosinophilic enteritis	27.3-4	
		Chemotherapy induced	30.12	
		Acute GI-GvHD	31.7	
		SLE	29.13	
	Edema	Portal hypertension	21.13	V23-02
	Mucosal hemorrhage	Radiation enteritis	22.11	
		Crohn's disease	25.9	
	Multiple red and white spots	Systemic mastocytosis	50.13	
Erythema	Patchy	NSAIDs	10.6c	V30-03
		Portal hypertension	21.13	
		Acute GI-GVHD	31.2	
	Distant tumor	Carcinoma involving mesentery	34.3	V34-01
	Fibrosis	Radiation enteritis	5.23b	
	Patchy villous atrophy	Eosinophilic enteritis	27.03	
	White villi	Eosinophilic enteritis	27.5	

	White villi, infiltration	Lymphoma EATL	26.7	
	Edema	Food allergy	38.1b	
	White villi	Healing IgA vasculitis	29.6c	
Aphthae		Crohn's	24.4	V24-01
		Systemic lupus erythematosus	29.12	
	Nodules	Intestinal spirochetosis	28.6	V28-03
Erosion		Crohn's	10.15b	V24-03
		NSAIDs	30.2-3	
		Intussusception	38.15	
	Villous atrophy	Celiac disease		V26-04
		Autoimmune enteropathy	26.9	
	Exudation	Acute GI-GVHD	31.4	
	Bleeding	Acute GI-GVHD	31.5	
	Mucosal hemorrhage	Collagenosis	22.5	
Ulcer	Aphthous	Crohn's	24.8	V24-03
		NSAIDs	38.3c	
			30.4	
	Fissural	Crohn's	24.7	
	Linear	Crohn's	25.7	
			41.7	
		Eosinophilic enteritis	27.9 g	
		Tuberculosis	28.5	
		IgA vasculitis	29.7	
		Behçet's	29.8	
		NSAIDs	30.5	
	Crater like	Crohn's	24.10	
		Eosinophilic enteritis	27.5c	
		Peptic duodenal ulcer	44.23	V44-07
		APC ulcer	39-29	
		Mucosectomy ulcer	39.30	
		Metastatic gastric cancer	34.28	
	(Semi)circular	Anastomotic ulcer	39.14	V39-09
		NSAIDs	30.5-7	
		NSAIDs stricture	30.7-8	V16-02
		Chronic nonspecific multiple ulcer of the small intestine	32.7-8	V32-01
		Meckel's diverticulum	20.11	V38-06
		EATL	41.15	
	Petechiae	Crohn's	2.2	V25-01
	Edematous stenosis	Crohn's disease	25.8	V24-02
	Second lumen	Diverticulitis	20.7	
		Meckel's diverticulitis	20.12	
		Anastomotic ulcer	39.12	V39-08
	Bleeding	Dieulafoy's ulcer	21.6	
		Peptic ulcer		V44-08
		Ischemic enteritis	21.15-16	V14-02
	Punched out	Cytomegalovirus	28.8	V28-04
		Cocaine abuse	30.14	
		Sarcoidosis	29.4b	
		PPI therapy	30.15	
		Biopsy ulcer	39.28	V39-10
	Necrotic	CMV/acute GI GvHD	31.11	
		Vasculitis	29.10	
	Multiple, diffuse	Amyloidosis	29.1	V29-01
		Vasculitis	29.10	

		Crohn's	25.8b	V24-02
		Acute GI-GvHD	31.8	
	With villous atrophy	Refractory sprue type II	26.5	V26-06
		Ulcerative jejunoileitis	41.14	V26-07-08
		EATL	34.19	V34-12
		Idiopathic chronic non-granulomatous jejunitis	50.11a	V26-11
		Ischemic enteritis	21.15	V21-08
	On submucosal tumor	Neurofibroma	33.32	
		GIST		V34-08
		Lipoma	33.29	V33-09
		Fibrolipoma	33.31	V33-10
		Amyloid tumor	29.3	
Diverticula	Proximal	Juxtapapillary diverticulum	20.1	
		Duodenal diverticula	20.2	V20-01
		Jejunal diverticulum	20.3	
	Multiple	Jejunal diverticulosis	20.4	V20-02
	Bleeding	Jejunal diverticulum	20.6	V20-03
	Distal	Meckel's diverticulum	20.8-9	V20-05
	Ulcerated, multiple	Diverticulitis	20.7	
	Ulcerated, single	Meckel's diverticulum, adult	20.11-12	
		Meckel's diverticulum, child	38.11	V38.06
	Gastric heterotopia	Meckel's diverticulum	20.10	V20-04
	Inverted	Meckel's diverticulum	20.13	V20-06
Scar		Ileocolostomy	39.17-19	V39-04
Nodules		Sarcoidosis	29.4a	
	Lymph follicular	Normal terminal ileum	19.18a	V19-17
	Ileum	Lymphoid hyperplasia	33.10a	V19-19
		Crohn's	24.11	
		HIV, CMV	28.12	
		Mycobacteriosis	33.10b	
	Entire small bowel	CVID	29.15	
		IgA deficiency	38.18	
		Giardiasis	28.15	
		Intestinal spirochetosis	28.6	V28-03
		IPSID	28.7	
Polyps	Sessile	Tubular adenoma	15.8	V15-02
	Flat, white	FAP	35.6	V35-03
		Sporadic duodenal adenoma	33.18b	V33-03
	Sessile, bleeding	Capillary hemangioma	33.21	V16-01
	Villous	Adenoma	10.14d	V33-05
	Ulcerated	Suture granuloma	33.2	
	Pedunculated	Peutz-Jeghers polyposis	10–12c	
	Large	Peutz-Jeghers polyposis	38.5	V38-03
	Medium	Peutz-Jeghers polyposis	10.13b	
		Juvenile polyp	33.13	
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		Lynch syndrome	37.1b	
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	With ulcer	Juvenile polyposis	37.3	V37-02
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	Bleeding	Tubular adenoma	33.20	
	Probably pedunculated	Peutz-Jeghers polyposis	10.12d	V36-02
		Inflammatory pseudopolyp	33.3	
		Hamartomatous polyp	33.11-12	V33-02

	Multiple	Peutz-Jeghers polyposis	36.4	
	Various sizes	Peutz-Jeghers polyposis	10.13d	
	Large	Peutz-Jeghers polyposis	36.4	V36-01
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	Sessile and pedunculated	Sea blue histiocytosis	29.19	
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	Polypoid	Kaposi's sarcoma	34.8	
		MALT lymphoma	34.18	
		Pleiomorphic cell sarcoma	34.12	
	Duodenal bulb	Brunner's gland hyperplasia	33.8	
		Ectopic gastric mucosa	44.21-22	V44-06
	Cystic	Lymphangioma	33.16	
	Crater like	Metastatic gastric cancer	34.28	
		Endemic Kaposi's sarcoma	34.9	
	In Meckel's diverticulum	Ectopic gastric mucosa	20.10	V20-04
	Anastomotic	Suture granuloma	33.2	
Submucosal	*Soft,* yellow	Lipoma	33.28	V33-06
	Yellow, multiple	Intestinal lipomatosis	33.30	
	Ulcerated	Ulcerated lipoma	33-29	V33-09
	Yellow, cystic	Lymphatic cyst	33.23-25	V19-16
	Dark, vascular	Hemangioma	2.4	V33-07
			33.21–22	
		Blue rubber bleb nevus	21.7	V38-04
		Varices	21.10-12	V21-06/07
			23.6	
		Venectasia	21.9	V19-16
	Firm			
	Whitish	GIST	41.3	V34-07
		Leiomyoma	33.27	V33-08
	Reddish	GIST	34.7	V34-08
	Dark, ulcerated	Amyloid tumor	29.3	
	Ulcerated	Neurofibroma	33.32	
	Bleeding ulcer	Fibrolipoma	33.31	V33-10
	Pathological vessels	Neuroendocrine tumor	05.8	V34-03
	Umbilicated, multiple	Neuroendocrine tumor	34.5	V34-04
Exophytic	Stenosing	Adenocarcinoma	34.3-4	V34-01
	Ulcerated	Adenocarcinoma	34.2	V40-05
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	White villi	Neuroendocrine carcinoma	34.6	
	Central depression	Mantle cell lymphoma	34.13	V34-09
	Eccentric nodules	Follicular lymphoma	34.14	V34-10
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	Ulcerated	Angiosarcoma	34.10	
	Ulcerated, multiple	Epithelioid angiosarcoma	34.11	
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		Kaposi's sarcoma	34.8	

Infiltrating	Ulcerated	Lymphoma	38.13	
		Centroblastic lymphoma	34.17	V40-07
		EATL	2.7	V34-12
			34.19	
	Ulcer, focal erythema	Pancreatic head carcinoma	34.30	
	White villi	MALT lymphoma	34.18	
	Erythema, white villi	Metastatic lung cancer	34.27	
	Vesicular	Follicular lymphoma	34.15	V34-10

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Video Capsule Endoscopy: A Reference Guide and Atlas

Video 02-01: Flashing capsules. PillCamSB3, EndoCapsule ECS10, MiroCam, OMOM capsule, CapsoCam, PillCamEso2, PillCamColon2 (Niehls Kurniawan and Martin Keuchel; Hamburg, Germany)

Video 05-01: Software demonstration. Rapid 8, Rapid 8 software (Carola Pflüger; Hamburg, Germany)

Video 05-02: EndoCapsule 3D localization. EndoCapsule software (Stefanie Bosselmann; Hamburg, Germany)

Video 05-03: 10 mm pellet in porcine small intestine. PillCamSB1 (Florian Gräpler; Tübingen, Germany)

Video 05-04: 2 mm pellet in porcine small intestine. PillCamSB1 (Florian Gräpler; Tübingen, Germany)

Video 05-05: PillCamSB2 (sized 11 × 26 mm) seen by second PillCam. PillCamSB2 (Martin Keuchel; Tübingen, Germany)

Video 05-06: Angiectasia, native and with FICE1. PillCamSB2 (Peter Baltes; Hamburg, Germany)

Video 05-07: Adenocarcinoma: native image, structure enhancement, FICE 1-3, blue mode. PillCamSB2 (Martin Keuchel; Hamburg, Germany)

Video 05-08: Active bleeding in the jejunum. EndoCapsuleEC1 (Martin Keuchel; Hamburg, Germany)

Video 05-09: Artifact–endoscope light. PillCamSB2 (Peter Baltes; Hamburg, Germany)

Video 05-10: Air bubble mimicking double lumen. PillCamSB2 (Peter Baltes; Hamburg, Germany)

Video 05-11: Large air bubble. PillCamSB2 (Peter Baltes; Hamburg, Germany)

Video 05-12: Small air bubbles obscuring mucosal visualization. PillCamSB2 (Peter Baltes; Hamburg, Germany)

Video 05-13: Coffee grounds in ileum. PillCamSB2 (Peter Baltes; Hamburg, Germany)

Video 07-01: Swallowing problem with endoscopic capsule placement. PillCamSB3, gastroscopy, AdvanCE delivery device (Uwe Matsui; Hamburg, Germany)

Video 07-02: Procedure of endoscopic capsule placement with AdvanCE delivery device. PillCamSB2, gastroscopy, AdvanCE delivery device (Martin Keuchel; Hamburg, Germany)

Video 07-03: Capsule transport from stomach to duodenum with Dormia basket. Gastroscopy, Dormia basket (Martin Keuchel; Hamburg, Germany)

Video 09-01: Intact M2A Patency capsule. PillCamSB1, M2A Patency capsule (Martin Keuchel; Hamburg, Germany)

Video 09-02: Coating of dissolved PillCam patency capsule in a patient with stenosing adenocarcinoma. PillCamSB2, PillCam patency (Martin Keuchel; Hamburg, Germany)

Video 13-01: Principle of double balloon enteroscopy. Courtesy of FujiFilm. Düsseldorf, Germany

Video 13-02: Crohn's stricture, hydrostatic balloon dilation. Antegrade double balloon enteroscope, dilation balloon (Hironori Yamamoto; Tochigi, Japan)

Video 13-03: Jejunal adenoma, endoscopic resection. EndoCapsuleEC1, antegrade double balloon enteroscopy, chromoendoscopy, endoscopic mucosal resection (Hironori Yamamoto; Tochigi, Japan)
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Video 14-02: Bleeding Dieulafoy´s lesion. PillCamSB3, Single balloon enteroscope, injection, clips. (Ingo Steinbrück, Christoph Ruether; Hamburg, Germany
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Video 21-01: Single medium-sized angiectasia in the ileum. PillCamSB2 (Martin Keuchel; Hamburg, Germany)
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Video 21-05: HHT hereditary hemorrhagic telangiectasias. PillCamSB3 (Ellen Nötzel; Berlin, Germany)
Video 21-06: Small varix with erosion, angiectasias. PillCamSB2 (Uwe Matsui; Hamburg, Germany)
Video 21-07: Large varix with defect and bleeding. PillCamSB2 (Jörg Albert, Frankfurt/Main, Germany)
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A Short History of the Gastrointestinal Capsule

1

Gavriel J. Iddan

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The origin of work on the gastrointestinal (GI) capsule can be traced back to 1981. At that time, I was on a sabbatical leave from my work as an electro-optical engineer at Rafael, a government defense lab in Israel, and was working in the United States for a medical instrument company in Boston, Massachusetts. A gastroenterologist friend, Prof. Eitan Scapa, explained to me some of the shortcomings of the fiber bundle endoscope, especially its rigidity and its inability to view the small intestine. At that time, I had no idea as to how to solve these intriguing and interesting problems.

Subsequently, small charge-coupled device (CCD) imagers had been developed and made available (mainly in Japan) for use in handheld video cameras. The endoscope manufacturers were quick to incorporate them into the endoscope, replacing the fiber bundle that was used for image transmission and making the device much more flexible. Nevertheless, there was still no satisfactory solution to the problem of how to view the small intestine.

My gastroenterologist friend kept querying me about ways to solve the problem, and while I was on another sabbatical in 1991, I started to think about the possibility of separating the CCD head from the endoscope, leaving it connected via an umbilical cable. It was explained to me that this method would be impossible because the cable would need to be about 5 m long, too long to be safely pulled out. Also, the process might last a few hours, and the endoscope would have to stay inside the patient all this time.

At that point, I intuitively asked, "Why not cut the CCD head from the endoscope and attach a minitransmitter to it, letting the head move free of any physical connection?" The chance of solving the problem then seemed more realistic, and in 1992 I started spending more time on the new idea. I realized, however, that the task I was facing was very far from having a solution.

Consultation with a CCD expert was very discouraging, as simple calculations indicated that a CCD camera head would be able to operate for only about 10 min on miniature batteries. It was also explained to me that because not too

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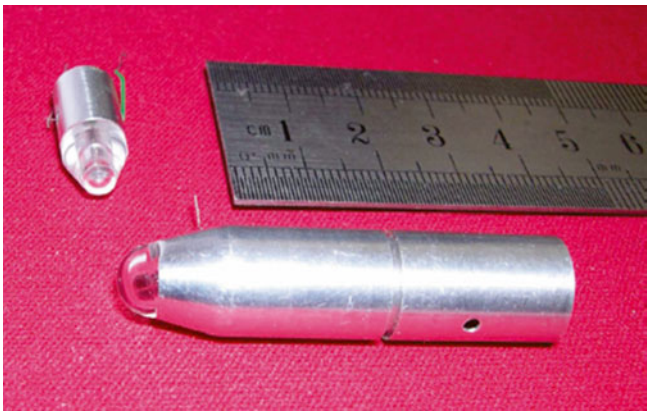

Fig. 1.1 Early, noningestible wired devices with a ¼-in. CCD used for testing the optics and the illumination

many pathologies are found in the small intestine, such a device would have only limited demand.

Because I was sure that the miniaturization problem would be solved in due time, I decided to continue my work on this very interesting and challenging problem and decided to focus on three major problems.

First, I figured that to avoid window contamination and obscuration, the optics would have to be designed in a way that would guarantee constant rubbing of the tissue on an ogive-shaped window to facilitate contact imaging and ensure self-wiping of the transparent window. A talented optical designer came up with a fine solution, and the prototype was built in 1993 (Fig. 1.1); a ¼-in. conventional CCD was used to test the optics and the results were good.

The next problem, long viewing hours, was solved by separating the system into three components: the *capsule*, containing the imager and transmitter; the *recorder*, containing an antenna array receiver and recording medium; and the *workstation*, incorporating the reader, processing software, and monitor. Simultaneously, we performed experiments to find the wavelength and power level required for wireless transmission of video through biologic tissue. (The experiments were done on a defrosted chicken bought in a nearby supermarket.) It seemed that we were on the right track but for one major obstacle—the power required by the CCD.

While casually reading a photonics magazine, I came upon an article written by Eric Fossum, a scientist at the Jet Propulsion Laboratory (Pasadena, California, USA), describing a new type of imager, the active pixel sensor (APS), which can be integrated on a single chip. Even more interesting, it was claimed to consume only 1 % of the power required by an equivalent CCD imager.

The APS was exactly what I was hoping for, and light appeared at the end of the tunnel. We submitted a patent application on 17 January 1994 and began to search for investment funds and start full-time work on the project. It was difficult to find support because investors considered the project "science fiction, an Asimov-type adventure." During my search for investment, in 1995 I came upon a small company making miniature CCD cameras for medical applications and tried to interest its manager in the new video pill; the manager, Mr. Gavriel Meron, became excited and tried to raise money from his board but was refused.

At the same time, I succeeded in establishing a new start-up, 3DV Systems, in the area of three-dimensional (3-D) imaging, using funds from a new investment body, Rafael Development Corporation (RDC) Ltd.—an indication that the high-tech market was on the rise. While working at the imaging start-up, in 1997 we were awarded the first US patent on a video capsule: US 5604531, "In vivo video camera system." The patent approval triggered action by RDC, and Mr. Meron joined RDC and incorporated a new start-up, Given Imaging Ltd. I served simultaneously as a vice president at the 3-D imaging start-up and as a consultant at Given Imaging. With initial funds available, expert workers were hired and capsule development went on at full steam; Mr. Meron was able to attract more investors in Israel and abroad.

At a 1997 gastroenterology conference, Mr. Meron met Prof. C. Paul Swain from London, and they were both surprised to find that they were working independently on related subjects, as Professor Swain and his team were aiming at a wireless gastric camera. An agreement of cooperation resulted, and Prof. Swain joined the Given team and contributed extensively to the development and to the animal and clinical experiments. Professor Swain described his group's efforts in the historical review published in our article in *Gastrointestinal Endoscopy Clinics of North America* [1].

Work progressed rapidly under the supervision of Dr. Arkady Glukhovsky, who was at that time the research and development manager. A CMOS (complementary metal oxide semiconductor) camera chip was designed by Eric Fossum to specifications written by Mr. Dov Avni, our video expert, and it was manufactured at Tower Semiconductor (Figs. 1.2 and 1.3). In October 1999, at the private clinic of Prof. E. Scapa near Tel Aviv, the first real capsule was swallowed by Professor Swain, who insisted on being the first person to swallow the capsule. After some initial difficulties, clear images were received. A bottle of wine was opened, and the video capsule turned into reality.

As a result of our initial success, more funds became available and work accelerated. An article published in *Nature* in May 2000 described the new capsule [2]. Experiments on consenting patients started in Israel, Europe, and the United States, with encouraging results. By August 2001, Given Imaging was ready for an initial public offering (IPO) on the NASDAQ Stock Market, but this was delayed by the September 11 World Trade Center attacks. A couple of weeks later, Given's IPO was the first

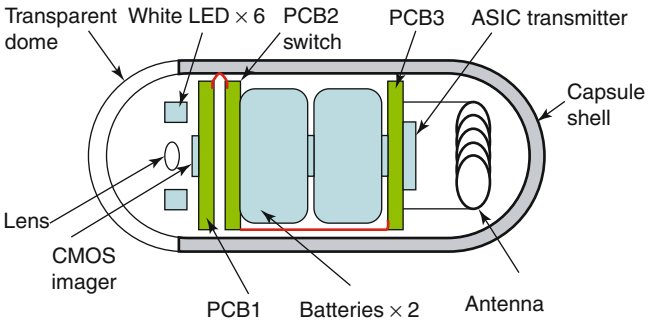

Fig. 1.2 A schematic view of the capsule and its components

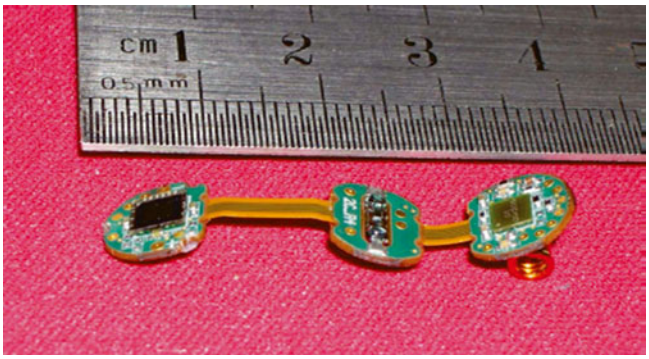

Fig. 1.3 Photo showing the three printed circuit boards: the left one holds the complementary metal oxide semiconductor (CMOS) imager, the central one holds the reed switch, and the right one holds the application-specific integrated circuit (ASIC) transmitter on the top and the antenna on the bottom

issue after the tower disaster. During the past decade, the video capsule has become a standard diagnostic tool used by gastroenterologists throughout the world; it is sold in more than 80 countries, and more than 1.8 million capsules have been ingested.

Since its introduction, researchers and practicing physicians have published more than 1,700 articles in professional journals describing all aspects of capsule usage. The fast-growing number of articles indicates that interest in the capsule is steadily growing.

The new capsule generation has an extended range of indications, and it is now considered as a leading diagnostic tool for intestinal bleeding, Crohn's disease, and other gastrointestinal disorders, as described elsewhere in this text. The new capsules incorporate a larger field of view, higher image quality, and a higher frame rate, all leading to superior performance and enabling a shorter viewing time. The new Data Recorder has new features such as a small monitor screen for real-time viewing and a two-way communication channel.

Most important, the colon capsule was introduced for cases in which conventional colonoscopy should be avoided. It is now being used mainly in Europe and has been submitted for approval by the US Food and Drug Administration (FDA).

At Given, we continue to develop better capsules with higher performance and new sensing capabilities that will enable the physician in the future to maneuver the capsule and control it from the outside of the body, thus improving the quality and increasing the speed of GI diagnostics.

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Fields of Application

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and David E. Fleischer

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Video capsule endoscopy (VCE) is an established endoscopic modality that allows remote examination (without intubation) of almost the whole of the gastrointestinal (GI) tract. The first device was developed to examine the small bowel, previously inaccessible to endoscopic examination, for which it is now a first-line investigative modality. Most commonly, small bowel VCE is used in patients with suspected bleeding or to identify evidence of active Crohn's disease (in patients with or without a prior history of the disease). Conventionally, VCE is undertaken after upper and lower gastrointestinal flexible endoscopy has failed to make a diagnosis. (Small bowel radiology or a patency capsule test should be considered prior to VCE to minimize the risk of capsule retention in patients at high risk of strictures, such as those with Crohn's disease, a long history of ingestion of nonsteroidal anti-inflammatory drugs [NSAIDs], or obstructive symptoms.) VCE may also be used in patients with celiac disease, polyposis syndromes, and other small bowel disorders. Since the advent of small bowel capsule endoscopy (SBCE), dedicated esophageal and colon capsule endoscopes have expanded the fields of application to include the investigation of the upper and lower GI tract as well as midgut disorders. Esophageal capsule endoscopy (ECE) may be used to diagnose esophagitis, Barrett's esophagus, and varices, but it cannot be relied on to identify gastroduodenal disease. Colon capsule endoscopy (CCE) offers an alternative to conventional colonoscopy for symptomatic patients, and a possible role in colon cancer screening is intriguing. Current research is already addressing the possibility of controlling capsule movement and developing capsules that allow tissue sampling and the administration of therapy.

2.1 Small Bowel Capsule Endoscopy

2.1.1 Obscure Gastrointestinal Bleeding

Obscure GI bleeding is the commonest indication for small bowel capsule endoscopy (SBCE) [1–5]. The term is conventionally used when esophagogastroduodenoscopy

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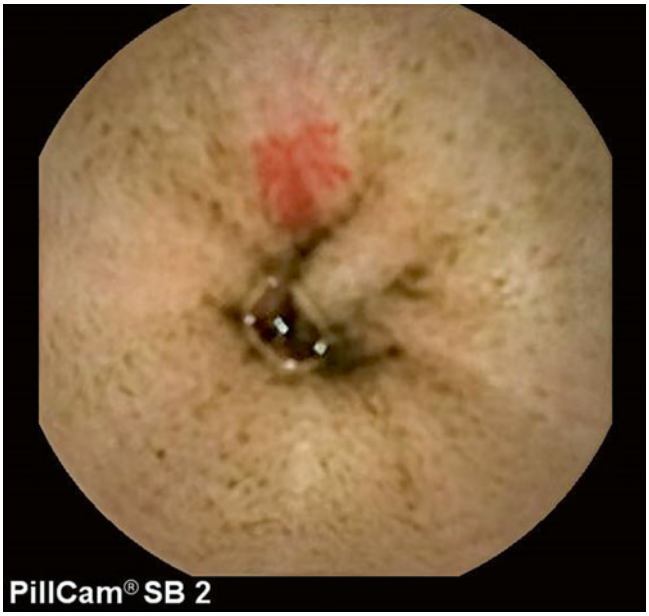

Fig. 2.1 Angioectasia

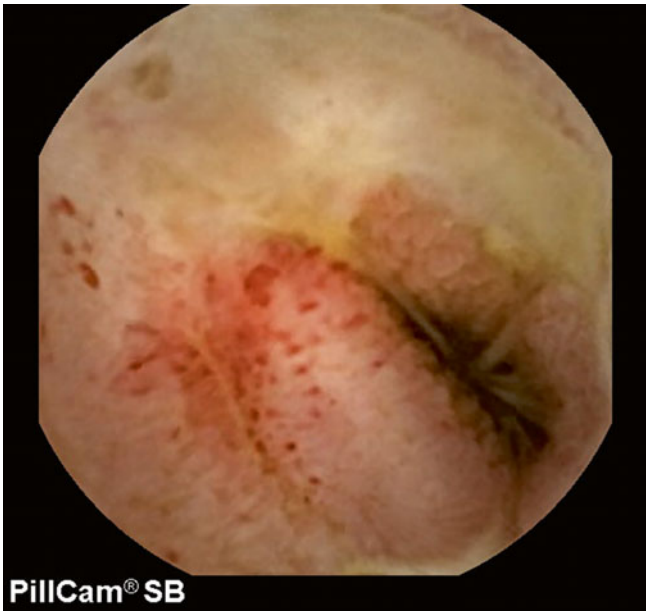

Fig. 2.2 Extensive ulceration and multiple petechiae caused by Crohn's disease

and colonoscopy have failed to identify a clear cause. Small bowel bleeding may be overt, manifesting with melena or hematochezia, or it may be occult, causing anemia. SBCE has a higher sensitivity than small bowel barium contrast radiology, small bowel CT, MRI, push enteroscopy, or angiography [6–11]. SBCE is recognized as a first-line investigative modality for obscure GI bleeding, with a diagnostic yield of 42–60 % [12, 13]. This yield is similar to that of double balloon enteroscopy [1, 4, 14, 15], which has the advantage of allowing biopsy or therapy, but the noninterventional nature and simplicity of SBCE mean that most clinicians use it to select patients (and target the lesion identified) for interventional endoscopy (see Chap. 41). Yield is improved if the procedure is performed during the episode of bleeding or as close to it as possible [16, 17]. Repeat VCE has a further yield of 35–75 % [18, 19], suggesting that, as with other modalities, lesions can be missed.

The reason that SBCE has greater sensitivity than other diagnostic modalities is that most lesions identified are flat vascular or mucosal lesions. Angioectasia—small, usually well-demarcated venous abnormalities that are often multiple—is the most commonly detected abnormality (Fig. 2.1) (see Chap. 21). Inflammatory lesions (ulcers and erosions) are also common findings [12, 13]. These may be due to Crohn's disease (Fig. 2.2), which occasionally presents with anemia or bleeding. However, an NSAID enteropathy may be indistinguishable from Crohn's disease and is probably underrecognized (Fig. 2.3) [20]. Vasculitis (Chap. 29), ischemia (Chap. 21) may cause small bowel ulcers, and infections like tuberculosis and cytomegalovirus (Chap. 28) should be considered in the appropriate clinical context. Finally, minor mucosal breaks are recognized in 7–40 % of healthy volunteers [21, 22].

Small bowel tumors are the cause in about 4–10 % of patients with obscure gastrointestinal bleeding [12, 23, 24]. In addition to benign tumors such as hemangiomas (Fig. 2.4), these may include adenocarcinomas and gastrointestinal stromal tumors, neuroendocrine tumors, lymphoma, and metastases (particularly from melanoma (Fig. 2.5) and from breast, lung, and renal primary malignancies).

Less common causes include Meckel's or other diverticula, small bowel varices, and aortoenteric fistulae.

2.1.2 Suspected Active Crohn's Disease

VCE may be used to diagnose Crohn's disease de novo or to assess disease activity in patients known to have Crohn's disease. Meta-analyses suggest that VCE has a greater sensitivity in detecting inflammatory activity in both groups of patients than small bowel barium contrast studies, small bowel CT, push enteroscopy, and ileocolonoscopy [25, 26]. Fewer studies compare VCE with small bowel MRI, but available data suggest that they are equivalent. In clinical practice, the two studies are complementary: VCE detects early mucosal changes, whereas MRI is useful in assessing more established transmural disease and its complications without the need for irradiation or the risk of capsule retention. VCE may be at least as effective as ileocolonoscopy in detecting early postoperative relapse, is better tolerated [27–29], and can be used to reclassify a proportion of patients with unclassified inflammatory bowel disease as having

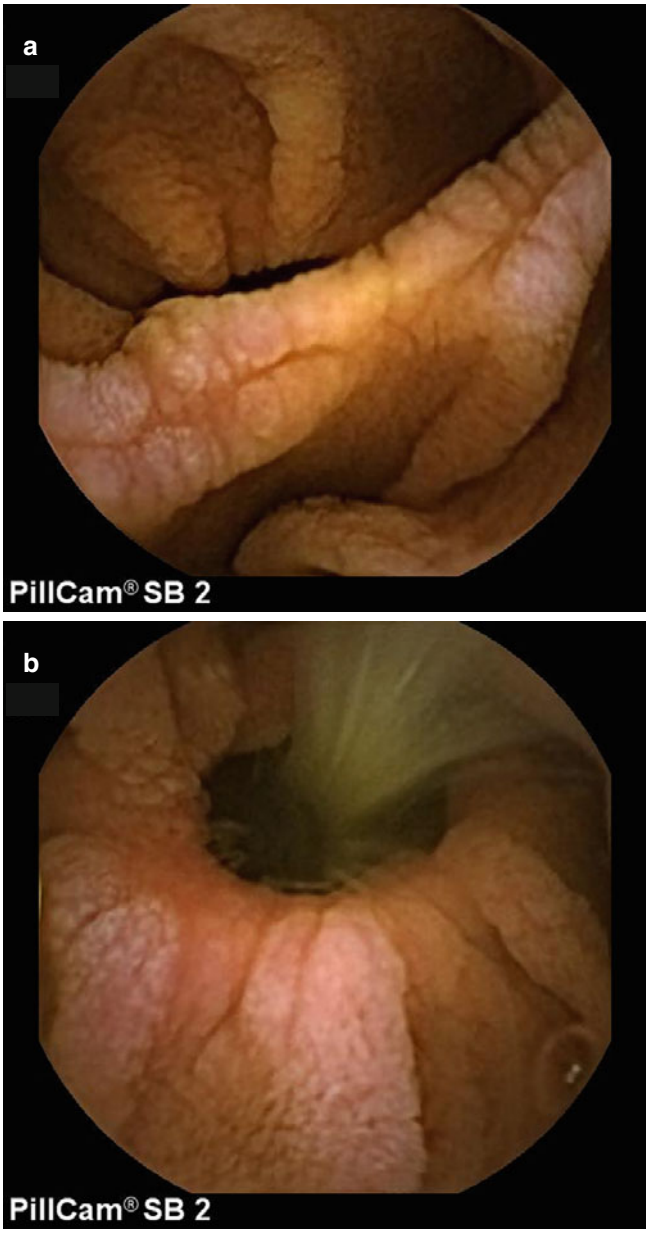

Fig. 2.3 (**a**) Villous atrophy with notching of folds and a linear mucosal break along the tip of a fold. (**b**) Stenosis with denuded mucosa and atrophic villi caused by ingestion of nonsteroidal anti-inflammatory drugs (NSAIDs)

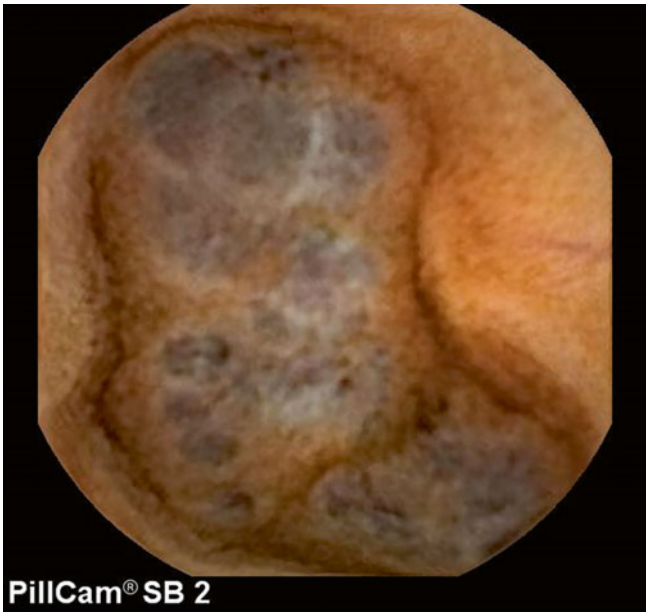

Fig. 2.4 Large hemangioma

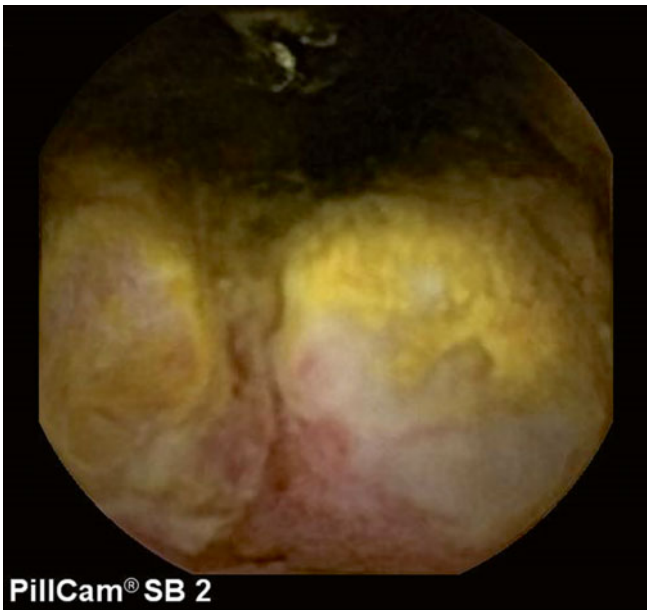

Fig. 2.5 Ulcerated tumor mass with slight blue pigmentation owing to metastatic malignant melanoma

Crohn's disease by demonstrating small bowel inflammation [30–33]. A possible role in assessing mucosal healing after treatment requires further investigation [34].

The main concern about VCE is the risk of capsule retention, which is between 5 and 13 % in patients known to have Crohn's disease, although retention in those being investigated for suspected Crohn's disease and obscure GI bleeding is similar at about 1 % [4, 35, 36]. Existing radiologic methods do not always exclude the possibility of short strictures [1, 35]. Use of the swallowable PillCam® patency device (Given Imaging, Yoqneam, Israel), which contains a radiofrequency tag, is effective in predicting safe passage of the capsule in the absence of a radiofrequency signal detected 30 h after ingestion (Chap. 9) [35]. Published guidelines recommend that prior investigation of the small bowel using the PillCam® patency device or alternative radiologic methods should be considered for patients with known or suspected Crohn's disease, particularly those with significant abdominal pain [1, 4, 37].

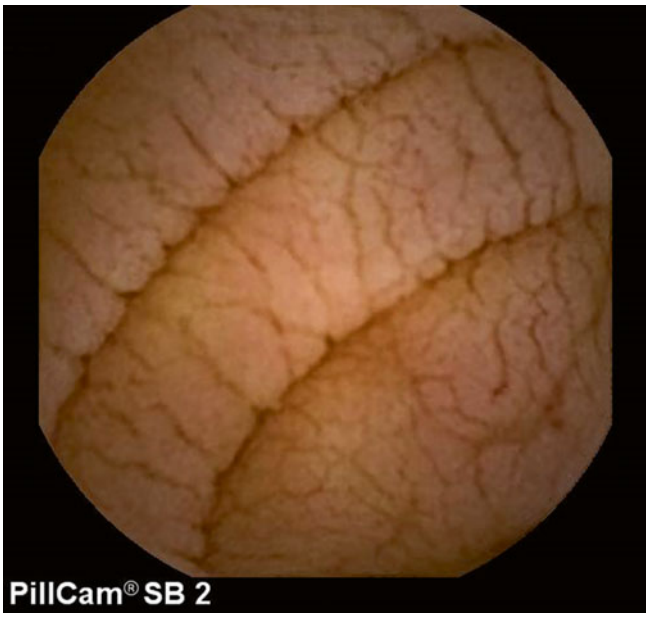

Fig. 2.6 Villous atrophy, with scalloping and notching of folds and a mosaic pattern due to celiac disease

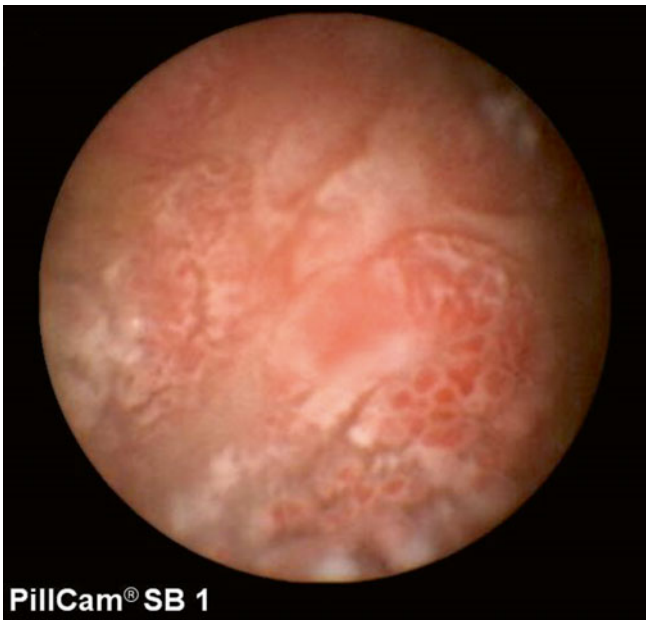

Fig. 2.7 Enteropathy-associated T-cell lymphoma complicating celiac disease. Note the absence of normal villous surface, nodularity, and ulceration

2.1.3 Celiac Disease

Meta-analysis suggests that VCE has a sensitivity of 89 % in making a new diagnosis of celiac disease, recognized by reduced and scalloped folds, a mosaic pattern, and micronodularity of the mucosa (Fig. 2.6) [38, 39]. Clearly, it does not recognize those without villous atrophy (Marsh grades 1 and 2), and therefore, duodenal biopsy remains the gold standard for diagnosis. Because specificity is as high as 95 %, it would be reasonable to consider VCE as a diagnostic test in patients with raised tissue transglutaminase levels or endomysial antibody titers if they refuse to undergo esophagogastroduodenoscopy.

VCE may have a role in patients with antibody-negative villous atrophy, either providing supportive evidence of a diagnosis of celiac disease or identifying features more typical of Crohn's disease [40]. The main clinical use for VCE, however, is in patients who do not respond to a gluten-free diet or who relapse while on such a diet. The presence of villous atrophy alone may prompt a reassessment of dietary compliance or a consideration of immunosuppressive therapy. Severe inflammatory change, mucosal irregularity, or a tumor mass may suggest complications of ulcerative jejunitis, enteropathy-associated T-cell lymphoma (Fig. 2.7), or adenocarcinoma [40–42].

2.1.4 Polyposis Syndromes

VCE has a greater sensitivity in detecting the polyps of Peutz-Jeghers syndrome than barium studies; it also avoids irradiation and is preferred by patients [43, 44]. However, though VCE may identify more small polyps, it occasionally misses very large polyps identified by MRI, which is of equivalent sensitivity in diagnosing clinically significant lesions (>10 mm) and appears to be as well tolerated [45].

For duodenal surveillance in familial adenomatous polyposis (FAP), current models of forward-viewing VCE detect the ampulla of Vater in 8.6–43.6 % of patients, so this method should not replace side-viewing duodenoscopy [46–48]. Polyps distal to the ligament of Treitz are much more likely in the presence of duodenal polyposis, and VCE is more sensitive than radiologic methods in detecting these lesions [49–52], but existing data suggest that the development of small bowel malignancy distal to the duodenum is extremely rare, so there seems to be little justification for monitoring FAP polyps using VCE in the absence of symptoms [53].

2.1.5 Miscellaneous

2.1.5.1 Abdominal Pain

In patients with abdominal pain alone, the diagnostic yield of VCE is between 6 and 21.4 %; it is higher in patients with weight loss or raised acute-phase proteins [54–58]. VCE may be useful in patients whose pain is thought to be obstructive in origin but whose diagnosis remains elusive despite multiple investigations. Diagnoses were made in 26 % of a small series of 19 patients thought to have small bowel obstruction based on symptoms alone or with supportive radiologic abnormalities [59]. VCE in this setting would, of course, require that the patient understand that the diagnosis may lead to either endoscopic or surgical retrieval of a retained capsule.

2.1.5.2 Graft-Versus-Host Disease

Graft-versus-host disease (GVHD) causes anorexia, nausea, vomiting, abdominal pain, and diarrhea in stem cell transplant patients. It is important to distinguish GVHD from drug toxicity and infection, which may require a reduction in immunosuppression. The small bowel is the commonest site for GVHD. Small studies suggest that VCE may be at least as effective in diagnosing GVHD as upper or lower GI endoscopy and biopsy [60–62] and it may be better tolerated. Microbiologic investigation remains important, however, as it is not known whether VCE can distinguish between GVHD and viral infection.

2.1.5.3 Nonsteroidal Anti-inflammatory Drug (NSAID) Enteropathy

As many as a third of the population may be using an NSAID at any given time [63]. Yet ingestion of diclofenac for 2 weeks causes small bowel inflammation in 68 % of those who use it [64], all NSAIDs (including low-dose aspirin) may cause mucosal injury [20, 64], and NSAID enteropathy is evident in 50–60 % of patients on long-term treatment [65]. Mild changes include reddened folds, petechiae, denuded mucosa, and mucosal breaks, but bleeding and deep ulceration may occur, along with stenoses in the longer term [64, 65]. As with many over-the-counter medications, patients may fail to declare their use, and NSAID enteropathy is likely to be underreported [20]. The relevance to the clinician is that NSAID enteropathy may be clinically and endoscopically indistinguishable from Crohn's disease and other causes of small bowel inflammation, so it should be considered in the differential diagnosis regardless of the indication for VCE.

2.2 Esophageal Capsule Endoscopy

2.2.1 Gastroesophageal Reflux Disease

When compared with conventional upper GI endoscopy, esophageal capsule endoscopy (ECE) has shown a sensitivity of 50–89 % in identifying erosive esophagitis [66–69]. ECE is significantly better tolerated [69, 70]. Perhaps because reflux symptoms are not associated with visible injury in as many as 60 % of patients [71] and other gastroduodenal disease is not excluded, ECE has not been widely adopted for this indication.

2.2.2 Barrett's Esophagus

Barrett's esophagus is a premalignant condition affecting 5–15 % of patients with reflux symptoms [72]; of those affected, 0.12–0.5 % of patients per year develop esophageal adenocarcinoma [73]. Meta-analysis suggests that ECE has a sensitivity of 78 % in identifying Barrett's esophagus, compared with conventional upper GI endoscopy [74]. The excellent tolerability profile of ECE may make it suitable for screening for Barrett's esophagus, but it is not routinely recommended for average-risk individuals [75]. Endoscopic surveillance is performed using imaging techniques and histologic analysis not currently possible with ECE [76], so at present ECE is not a suitable alternative for those known to have Barrett's esophagus.

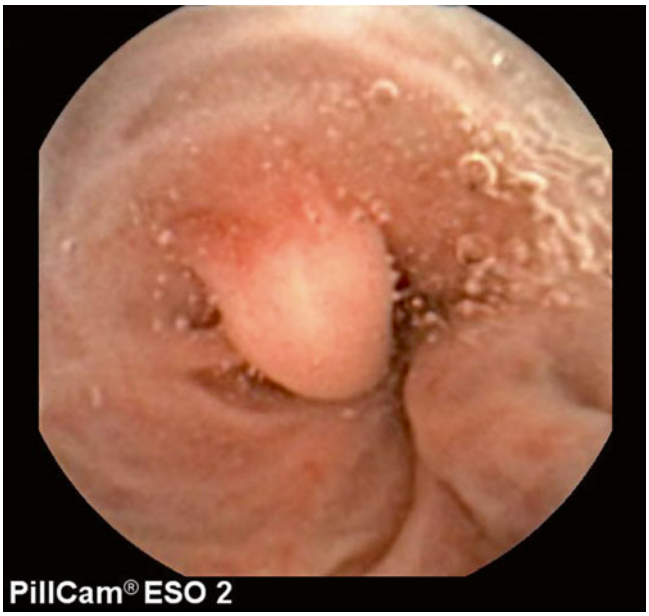

Fig. 2.8 Small varix, regenerative polyp, and scarring resulting from previous variceal banding in the esophagus

2.2.3 Esophageal Varices

Of patients with compensated cirrhosis, 40 % have esophagogastric varices, and there is a further 5–10 % incidence per year. Variceal bleeding is associated with a 40 % 1-year mortality rate. Because bleeding risk can be reduced with pharmacologic or endoscopic intervention, these patients undergo regular endoscopic screening to detect varices [77]. Meta-analysis shows that ECE has 83 % sensitivity in screening for varices (Fig. 2.8) when compared with conventional upper GI endoscopy [78], so it may prove to be a viable alternative in this setting.

2.3 Colon Capsule Endoscopy

2.3.1 Colorectal Neoplasia

The second generation of the colon capsule includes an improved angle of view of 172° and an adaptive frame rate that allows acquisition of images at a rate between 4 and 35 frames per second, dependent on the speed of travel of the capsule. Two large multicenter studies comparing second-generation colon capsule endoscopy (CCE) with conventional colonos-

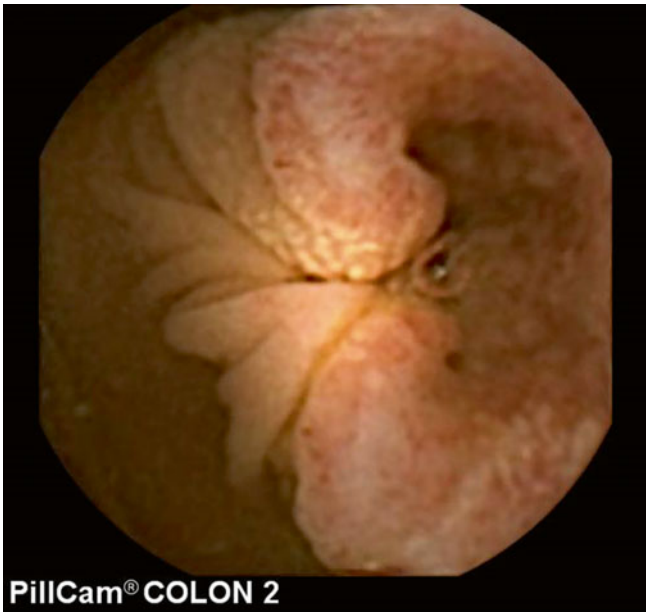

Fig. 2.9 Adenocarcinoma of the colon

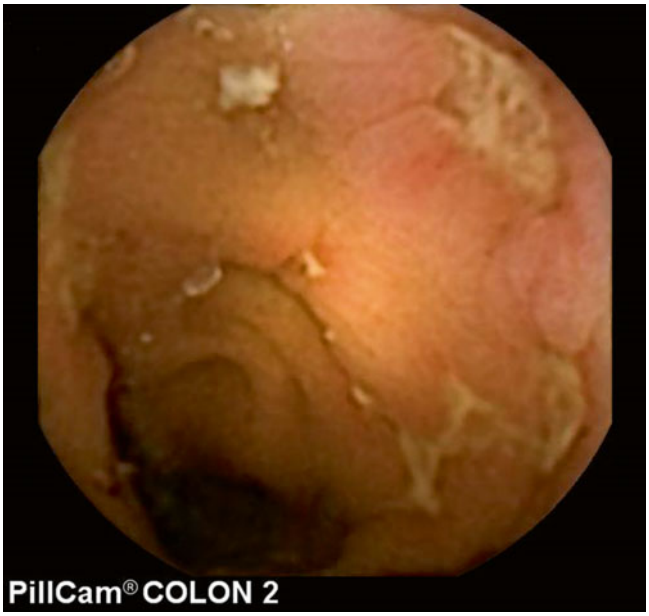

Fig. 2.10 Deep and serpiginous ulcers caused by Crohn's colitis

copy showed 89 and 84 % sensitivity in detecting polyps measuring at least 6 mm [79, 80]. Such outcomes are not dissimilar from results of CT colonography when compared with colonoscopy [81] or indeed from colonoscopy when compared to itself (in "back-to-back" or "tandem" studies) [82]. Current European guidelines consider that although colonoscopy remains preferable for individuals at high risk of colorectal cancer (because of alarm symptoms, a family or personal history of colorectal cancer, or both), CCE may be a viable alternative for average-risk individuals in whom colonoscopy is not possible (Fig. 2.9) [83]. An interesting study using mathematical modeling suggested that CCE would be more cost-effective than colonoscopy in population screening if it were associated with a 30 % better compliance rate [84]. However, this study is based on outcomes from trials that used the first-generation colon capsule, and the second-generation device represents a measurable improvement. Therefore, further studies are needed to clarify the role of CCE versus colonoscopy and CT colonography in symptomatic patients and in population screening.

2.3.2 Inflammatory Bowel Disease

A multicenter study comparing the first-generation colon capsule with colonoscopy in assessing the activity of ulcerative colitis found 89 % sensitivity, 75 % specificity, 93 % positive predictive values, and 65 % negative predictive values [85]. A second small study identified a significant correlation with colonoscopy in monitoring the severity and extent of inflammation [86]. It has been suggested that CCE thus may be useful in assessing treatment-induced mucosal healing, a predictor of better outcome (Fig. 2.10) [85]. However, even in a very small minority of patients with rectal sparing or patchy disease due to topical or other therapy [87], it seems likely that flexible sigmoidoscopy and biopsy usually will provide a relatively well-tolerated means of obtaining the required information.

2.4 Future Fields of Application of Capsule Endoscopy

The small bowel capsule was the first to be developed because of the clinical imperative: the inaccessibility of the small bowel meant that visualization of its mucosa was not possible without recourse to surgery. In addition, the size of the small bowel lumen lent itself to the possibility that a device small enough to be swallowed could visualize almost all of the surface area.

Visualization of the larger colonic lumen, part of which is obscured by the presence of haustral folds, was addressed by developing a double-headed capsule, which provides simultaneous antegrade and retrograde images that approach a 360° view.

The stomach provides a further challenge: optimal visualization requires distention such that the lumen develops substantial capacity. However, early studies have already demonstrated control of movement of a capsule in the stomach using magnets [88–90], and trials are under way to compare magnetically controllable VCE with conventional diagnostic upper gastrointestinal endoscopy.

It is now possible to examine the whole of the GI tract using VCE, and future technological developments may allow these devices to replace conventional upper GI

diagnostic endoscopy. Devices that allow sampling of fluid or tissue or that even can administer therapy are already on the horizon [91].

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