

Opportunities for the early detection and treatment of colon and rectal cancer in Hong Kong

MH Shiu

Colorectal cancer is the third most common cause of death due to cancer in Hong Kong. Most of these cancers are diagnosed at a late stage, when they are not amenable to curative treatment. Clinical trials have shown that screening for colorectal cancer using the faecal occult blood test can reduce death from colorectal cancer, but this test misses many cancers due to its inherent limitations. Double-contrast barium enema, flexible sigmoidoscopy and colonoscopy produce increasingly accurate results, at increasing cost. Selected population screening by faecal occult blood test, and targeted screening of persons at high risk using the more invasive tests, deserve consideration in Hong Kong. Amongst those at high risk are persons who have had rectal bleeding or colorectal adenomas, and those with one or more first degree relatives with colorectal cancer or adenoma. Early diagnosis would allow more effective treatment to be given, without the need for a colostomy, and give better odds for survival and quality of life.

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Introduction

Malignant neoplasms have surpassed heart and cerebrovascular diseases as the leading cause of death in Hong Kong. Among malignant neoplasms, death from colorectal cancer (CRC) ranks third, being less common than lung and liver cancer.¹ When considered as a single entity, cancer of the colon and rectum represent the second most common cancer for new cases reported to the government Cancer Registry in the year 1990.¹

The survival rate after treatment of CRC depends very much on the stage of the tumour when diagnosed. Diagnosis at an early stage enables effective, curative treatment, yielding a high survival rate. Late diagnosis often leads to ineffective treatment, morbidity and mortality, and with it a heavy drain on the resources of the patient, the family, and society.

Over the past three decades, many advances have come about in the early diagnosis and treatment of CRC. This paper reviews some of the recent concepts and techniques, and the possibility of applying these in Hong Kong.

Early diagnosis of symptomatic colorectal cancer

A high index of suspicion is the key to early diagnosis. Abdominal pain, rectal bleeding, anaemia, change of bowel habit and defaecation difficulties are all symptoms of colorectal cancer. However, most cancers present with only one of these symptoms, often in a subtle manner. For instance, a cancer of the caecum or ascending colon usually presents with occult anaemia, and no pain; a cancer of the transverse colon typically causes partial obstruction with intermittent central abdominal discomfort, often treated as dyspepsia until total obstruction sets in; a cancer of the rectum or sigmoid colon can cause rectal bleeding, a change in bowel habit or even tenesmus, but patients may attribute the symptoms to haemorrhoids. Haemorrhoids and cancer of the bowel often coexist. Many a patient diagnosed with advanced CRC tells of such early symptoms, sometimes for many months. It takes an

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Hong Kong Sanatorium and Hospital, Hong Kong

MH Shiu, MB, BS, FRCS

Correspondence to: Dr MH Shiu

educated patient and an astute clinician to arrive at the correct diagnosis by means of rectal examination, a barium enema or endoscopic examination.

Early diagnosis of asymptomatic colorectal cancer

Mass screening is theoretically the ideal method of finding all asymptomatic cancers in the population. A tremendous amount of time, manpower and financial resources are needed. Such population screening for gastric cancer has been successfully carried out in Japan where the high incidence of this disease affords a high yield, and sufficient resources are available to mount such a campaign. Neither the incidence of CRC nor the available resources can justify population screening in most countries, including Hong Kong. However, targeted screening can be effectively practised by all clinicians. By targeting the screening effort, the yield of early CRC can be increased, and the cost of diagnostic tests reduced. Two questions arise: which individuals are at most risk of developing CRC, and how can they be identified by one or more simple means? Once these individuals have been identified, follow-up tests are performed for surveillance reasons. The conditions in which such targeted screening or surveillance should be directed are discussed below.

Family history of colorectal cancer

A recent prospective study has shown that for individuals younger than 45 years with one or more affected first degree relatives, the relative risk of developing CRC is 5.3 times that of individuals without such affected relatives.² It is good clinical practice to counsel and screen the relatives of patients suffering from CRC. A careful enquiry into the family history can reveal the rare but very important hereditary types of CRC, such as familial adenomatous polyposis (FAP) syndrome, hereditary non-polyposis colon cancer syndrome (HNPCC), Gardner's syndrome (GS), Peutz-Jeghers disease, and familial juvenile polyposis. These are well-established heritable syndromes leading to CRC. There is now increasing evidence that many "sporadic" CRCs also have a genetic predisposition, and that screening efforts should be stratified according to the family history.³

Inflammatory bowel disease

Ulcerative colitis and Crohn's disease can predispose to the development of CRC.^{4,6} The risk of cancer amounts to 5% after 20 years and 12% after 25 years in patients with ulcerative colitis. The risk is less for

Crohn's disease. Endoscopic surveillance is needed in these patients.

Adenomatous polyps

Adenomas of the colon and rectum are the most important precursors of large bowel cancer. A patient who has had one or more colorectal adenomas removed is at risk of developing large bowel cancer. The prevalence of colorectal adenomas and CRC correlate in many countries.⁷ It has long been suspected that these adenomas are somehow related to the development of cancer. We see a wide spectrum of adenomas, with normal epithelium, low- to high-grade dysplastic changes, carcinoma in situ, and frankly invasive carcinoma within the adenoma—sometimes all in the same patient. Molecular genetic studies have shown evidence of step-wise events involving gene mutations, chromosomal deletions, and loss of tumour suppressor genes in the adenoma-carcinoma sequence.⁸ Cogent arguments can be made from this hypothesis that most, if not all CRCs develop from adenomas, and that if all adenomas can be removed, CRC cannot develop. However, evidence for this hypothesis was lacking until recently.

In a study published by the National Polyp Study Workgroup in the United States,⁹ 1418 patients underwent multiple sequential colonoscopy procedures, during which one or more adenomas were removed and followed for an average of 5.9 years. No symptomatic CRCs developed, but five early cancers (malignant polyps) were found and treated during the years of follow-up. All five patients were alive after surgical treatment. By comparison, in three control cohorts, 48, 43, and 21 patients developed invasive CRC, respectively. Control cohorts comprised historical control patients at the Mayo Clinic, Rochester, historical control patients at St. Mark's Hospital, London, and concurrent patients registered in the Surveillance, Epidemiology, and End Results (SEER) Program of the United States.¹⁰ The authors concluded that the absence of symptomatic cancer and the finding of only a few early polypoid cancers in the polypectomy group, support the view that colorectal adenomas progress to adenocarcinomas, and that removing them can prevent cancer. More importantly, surveillance colonoscopy also led to the diagnosis and effective treatment of early, asymptomatic CRC.

Studies in Hong Kong have also shown a close association between colorectal adenoma and cancer, as evidenced by a spectrum of colorectal neoplasms ranging from adenomas with mild to severe degrees of

dysplasia, carcinoma in situ, and invasive cancer. Chung et al reported their experience of 130 colorectal adenomas seen in the United Christian Hospital, Hong Kong, between 1986 and 1990.¹¹ Seventy-four patients had adenoma only, while 56 had adenoma plus an associated CRC. The presence of invasive cancer within an adenoma was frequently observed. The prevalence of colorectal adenomas in Hong Kong has also been confirmed by Cheung et al who studied the cause of rectal bleeding in patients over the age of 40 who attended the University of Hong Kong Proctology Clinic.¹² Of their patients, 9.1% and 10.3% had cancer or adenoma(s), respectively. These findings lend further support to the usefulness of screening tests for the early diagnosis of CRC and its precursor lesions. The question that remains is which screening test or tests should be used in a given setting?

Screening tests for colorectal cancer

Digital rectal examination

Digital rectal examination is regarded as part of a standard physical examination, rather than a screening test. It deserves emphasis because it is not always performed. Many a patient suffering from rectal cancer will confirm that their doctor did not perform a rectal examination or proctoscopy even though the patient's symptoms were ascribed to haemorrhoids.

Faecal occult blood test

The faecal occult blood test (FOBT) offers a simple, cheap, safe, and readily administered means of screening for colorectal neoplasms. The method has been used in more than 200 screening programmes, with favourable reports for the detection of adenomas and early cancers. Test kits can be used in doctor's clinics for immediate interpretation, or at home for examination by mail. The guaiac method is commonly used. A blue colour reaction due to phenolic oxidation of the guaiac by the peroxidase-like enzymatic action of iron-attached haeme indicates the presence of "occult blood". Several commercial kits are available, with modifications of the basic test. The test cross-reacts with meat myoglobin and gives a positive reaction to any peroxidase activity, including plant peroxidases. It can be blocked by the ingestion of large amounts of some raw vegetables or fruit, or by antioxidants such as vitamin C at a dose of 1 gm or more daily. The immunochemical method is not peroxidase-dependent and is more selective; it detects human haemoglobin, globin and the early degradation products of globin. The heme-porphyrin test broadly detects heme-derived porphyrins, including breakdown products such as de-ironed free heme or haemoprotein of human and animal origin.

When interpreting a positive or a negative FOBT, the clinician needs to consider the sensitivity and specificity

Table 1. Controlled trials of faecal occult blood screening for colorectal neoplasms

	Trial size	Cohort positivity rate (%)	Predictive value* (%)	<u>Dukes' A and B cancers</u>	
				<u>screened</u> (%)	<u>control</u> (%)
Göteborg, Sweden	27 000	1.9	22	65	33
Nottingham, England	150 000	2.1	53	90	40
New York, United States	22 000	1.7	30	65	33
Minnesota, United States	48 000	2.4	31	78	35
Fühnen, Denmark	62 000	1.0	58	81	55
* Predictive value for adenoma and cancer is the number of true-positives (neoplasms found) divided by the total number of all positive results					
Source: Winawer SJ, et al. Colorectal cancer. 1992. ⁴⁸					

of the test. A positive test will require further diagnostic studies to rule out causes other than colorectal neoplasm. The specificity of guaiac tests in screening trials has ranged from 90% to 98%, meaning that 2% to 10% have "false positive" tests, mostly due to non-neoplastic causes.¹³ Anxiety and expense are necessarily caused by the further diagnostic tests in these false-positive cases. A negative test cannot rule out the possible presence of a cancer because a CRC (or adenoma) does not always bleed. If a lesion bleeds, it may do so only intermittently. Prospective studies indicate that the sensitivity of guaiac-based tests is at best 30%, so that there is a 70% false-negative rate.¹³⁻¹⁵ In patients with CRC, less than one third of the cancers bleed 1.5 ml or more daily.¹⁴ This amount of bleeding in the bowel is at the lower border of sensitivity of some of the more sensitive guaiac test kits. It is a recognised inherent limitation of the test and does not detract from its value as a method of screening.

The usefulness of screening has been documented by at least five large-scale controlled trials of FOBT.¹⁶⁻²⁰ The trials showed test-positive rates of 1% to 4% and predictive values for colorectal adenoma and cancer of 22% to 58% (Table 1). Many more early-stage cancers (Dukes' A classification, 34% to 65%) were detected in the screened group, compared with only 8% of controls. An earlier trial showed a significant reduction in mortality from CRC to 30% in patients found to have cancer in the screened group, compared with a mortality of 50% in those in the control group.¹⁷ The Minnesota Colon Cancer Control Study²¹ recently published the results of screening 46 551 subjects in groups randomised to occult blood testing once a year, every two years, or no testing. The 13-year cumulative mortality per 1000 from CRC was 5.88 in the annually screened group, 8.33 in the biennially screened group, and 8.83 in the unscreened control group. A statistically significant reduction in mortality in the annually screened group was achieved compared with the control group. Useful and simple as the FOBT is for screening purposes, it fails to detect many cancers and adenomas. The simplicity of the method argues strongly in favour of its use in the screening of populations, but the method is not itself sufficiently sensitive for "targeted screening" in the doctor's clinic. Radiological or endoscopic tests should complement or replace the FOBT in this setting.

Barium enema

A properly performed double-contrast barium enema examination can detect 98% of all colonic adenomas and cancers greater than 15 mm in diameter, according to a prospective study by Fork et al.²² The sensitivity of polyp detection with double-contrast enemas was found to be

87%, compared with 59% for the single-contrast technique.²³ The error rate of single solid-column barium examination is very high in the detection of neoplasms,²⁴ and this technique is no longer used in most clinics. Most of the errors in barium enema examination occur in the sigmoid colon because of its redundancy and overlapping flexures. Barium-filled ileal loops, diverticular disease, and sometimes retained faeces add to the difficulties of obtaining optimal images. The caecum and proximal colon are usually well shown, but cancers can still be missed.²⁵ Barium enema examination is readily available in hospitals and radiology clinics, and its associated risk and cost are low. It represents one of the most useful methods for the "targeted screening" of patients in the clinic, provided double-contrast examinations of good quality are obtained, and the clinician recognises its limitations.

Sigmoidoscopy

The rigid sigmoidoscope has now been replaced by the flexible fiberoptic sigmoidoscope which can reach further into the bowel, and is much safer and more comfortable for the patient. It examines only 25 cm to 50 cm of bowel. Because a major proportion of cancers and adenomas occur in the sigmoid colon and rectum, the rigid sigmoidoscope was regarded as a valuable screening tool. Uncontrolled screening trials of rigid sigmoidoscopy in 21 150 patients performed in Minnesota suggested its use brought about a reduction in mortality.²⁶ Two similar case-control studies also suggested a reduction in mortality from CRC of 60% to 70%.^{27,28} No randomised clinical trial has been performed to prove that screening by sigmoidoscopy improves survival. Flexible fiberoptic sigmoidoscopy can be used together with the FOBT as a complementary screening method. Special equipment and experienced personnel are required.

Both the American Cancer Society and the American College of Physicians recommend that asymptomatic adults older than 50 years of age undergo screening by sigmoidoscopy to detect adenomas and early cancer, although the United States Preventive Services Task Force and the Canadian Task Force on the Periodic Health Examination do not support such screening because there have been no data from controlled studies that demonstrate a survival advantage.²⁸ The high cost of sigmoidoscopy as applied to population screening, and the lack of trained personnel are major concerns. For targeted screening, it is conceivable that primary care physicians and even nurse endoscopists could be trained to perform the procedure.²⁹ For the experienced endoscopist, the flexible sigmoidoscope is a very convenient tool for "targeted

screening", especially if it is combined with barium enema examination. The strength of sigmoidoscopy is in visualising the sigmoid colon which is the area most difficult to see well by barium enema. Biopsy of any lesions seen, and removal of polyps by endoscopic cauterly snare may also be performed at the time of sigmoidoscopy.

Colonoscopy

The entire colon and rectum can be visualised by colonoscopy in 95% of examinations performed by experienced endoscopists. However, in approximately 5% of examinations patient discomfort, presence of

convoluted curves and flexures, or pre-existing disease such as diverticulitis make it impossible to reach the caecum or clearly see all segments of the bowel. Barium enema examination is then used to complement an incomplete or unsatisfactory colonoscopy. Colonoscopy is much more accurate in the detection of small lesions (diameter 1.5 cm or smaller) than is double-contrast barium enema.²² Also, biopsy of lesions and therapeutic removal of polyps may be performed at the time of colonoscopy. Perforation of the bowel and major bleeding are potentially serious complications, occurring in fewer than 0.1% of examinations with experienced personnel.

Table 2. Guidelines for targeted screening of colorectal cancer

Condition	Recommended testing
Persistent vague central or low abdominal pain and/or change in bowel habit	FOBT and double-contrast barium enema
History of rectal bleeding or FOBT positive	Colonoscopy (preferred) or double-contrast barium enema
Patient with known colorectal adenoma or carcinoma	Initial colonoscopy to remove all adenomas if present; repeat every two to five years
First degree relative with colorectal cancer at age younger than 30 (or known FAP syndrome in family)	FAP or one of the other hereditary syndromes must be suspected;* flexible sigmoidoscopy and FOBT beginning at age 10 to 12 years
Three or more first degree relatives with colorectal cancer	HNPCC must be suspected; FOBT and colonoscopy every two years beginning at age 25 or at age five years younger than age at diagnosis of cancer, whichever is earlier
One or two first degree relatives with colorectal cancer	FOBT starting at age 35 to 40 or at age five years younger than age at diagnosis of cancer, whichever is earlier; colonoscopy every three to five years
Inflammatory bowel disease	Colonoscopy yearly or every two years beginning 8 to 10 years after onset of ulcerative colitis [†]
* Familial juvenile polyposis and Peutz-Jeghers syndrome can also cause colorectal cancer at a very young age	
[†] Patients with Crohn's disease are also at risk, although less so than those with ulcerative colitis	
FAP	familial adenomatous polyposis syndromes, including Gardner's syndrome, Turcot's syndrome and Oldfield's syndrome
FOBT	faecal occult blood test
HNPCC	hereditary non-polyposis colorectal cancer syndrome

Colonoscopy is not a tool for general population screening because of its expense and the need for considerable manpower and expertise. Rather, it is an important tool for definitive diagnosis when a screening test, such as FOBT, has yielded a positive result.

Targeted screening and surveillance

Colonoscopy is the most important method of targeted screening and surveillance for persons at very high risk of developing CRC. In these high-risk individuals, the periodic use of a definitive diagnostic test such as colonoscopy, rather than non-invasive screening, is justifiable because of a much higher likelihood of encountering cancer or adenomas which can be removed at the same time. While most authorities perform colonoscopy in these settings, there is less agreement on the frequency of colonoscopic examination. Cost and lack of endoscopic expertise may also curtail the application of colonoscopy. As an alternative, flexible sigmoidoscopy and double-contrast barium enema can be considered. Some guidelines are given below (Table 2).

Colorectal adenoma

Patients found to have an adenoma on barium enema or sigmoidoscopy examination should undergo colonoscopy to remove the lesion found, and any other adenomas not yet identified. A follow-up surveillance examination is also needed. These patients are at risk for the development of future adenomas, and of colorectal cancer if new adenomas are not removed. Morson's studies³⁰ suggested that it takes an average of two to three years to develop an adenoma larger than 1 cm in diameter, and seven to 10 years to develop a gross cancer. Observations from the United States National Polyp Study also indicate that the sequence of changes from normal colonic mucosa through adenoma formation and dysplasia to cancer takes 10 years.³¹ Repeat colonoscopy can therefore be performed three years later, but individuals may require more frequent follow-up colonoscopy if they have numerous adenomas, a carcinoma arising in an adenoma, incomplete removal of adenoma, or incomplete examination of the colon. Surveillance colonoscopy and polypectomy have proven to be highly effective in the prevention and early detection and treatment of CRC.⁹

Resected colorectal cancer

Patients who have had one CRC successfully resected are at risk of having or developing a second primary CRC (2% to 6%) and of adenomas (25% to 45%).³²

Hence, colonoscopy should be performed either before resection of the cancer, or, if this is not possible due to obstruction, within a short time after the operation. Repeat colonoscopy should be performed every two to five years to detect metachronous lesions, and more frequently if many adenomas are found.

Hereditary risk of colorectal cancer

A person who is found to have 25 to 30 or more adenomas of the colon must be suspected of having familial adenomatous polyposis (FAP). The development of CRC is a virtual certainty in individuals affected by this disorder. All known blood relatives should undergo surveillance examination commencing at 12 years of age. Sigmoidoscopy rather than colonoscopy is usually sufficient for diagnosis, and if diagnosed to have FAP, all affected subjects should undergo total colectomy at 17 to 18 years, or immediately if diagnosed at a later age. In the genetics laboratory, white blood cell DNA markers of the mutant gene of FAP on chromosome 5 can also be examined to determine if family members are affected.

Surveillance colonoscopy is also indicated in a family with hereditary non-polyposis colorectal cancer (HNPCC). An individual is considered likely to be affected if three or more first degree relatives suffer from CRC, colorectal and female genital cancer, or colon cancer diagnosed before age 40. A mutant gene on chromosome 18 is thought to be responsible, but DNA marker tests are as yet not available. Hereditary non-polyposis colorectal cancer may account for as many as 4% to 6% of colon cancers.³³ Although HNPCC is termed "non-polyposis" to distinguish it from FAP, subjects with HNPCC do develop adenomas at a frequency similar to patients who have "sporadic" CRCs. Cancer of the colon or rectum develops at a mean age of 41 years. Multiple cancers occur in 25%, and 60% of colonic tumours develop in the right colon.³⁴ Surveillance colonoscopy is begun at age 25, or at an age five years earlier than when the earliest colon cancer arose in the family before age 30. There is less agreement on how best to conduct surveillance of individuals who have one or two first degree relatives with CRC. The estimated life time risk for CRC is 12% to 18%³⁵ in these individuals. Annual FOBT should begin at age 35 to 40 years. Colonoscopy is also recommended every three to five years.³⁶

Inflammatory bowel disease

The risk of cancer in a patient suffering from ulcerative colitis is 20 times that of the general population after having had the disease for 20 years, and over 30 times after 30 years. Surveillance colonoscopy is performed annually or every alternate year at eight to 10

years after the onset of disease. The objective of colonoscopy is not only to detect cancer early, but also to obtain multiple-level mucosal biopsies to look for any dysplastic changes.^{37,38} Biopsy-confirmed cancer, or the detection of dysplasia with a mucosal mass may prompt the performance of total colectomy.³⁹

Treatment of early colorectal cancer

Detected early cancers of the colon and rectum present greater opportunities for cure. Modern treatment also allows better preservation of bowel function and quality of life, particularly for cancer of the rectum. A discussion of the management of specific types of early colorectal cancer follows.

Malignant colorectal polyps

When a polyp has been found to have carcinoma on microscopic examination after biopsy or snare polypectomy, the need for a formal bowel resection must be assessed. When only carcinoma-in-situ is found, polypectomy alone is curative. For invasive carcinoma, treatment depends on the gross and microscopic pathological characteristics of the polyp, and whether the polyp is pedunculated or sessile. For a pedunculated polyp, when the carcinoma is confined to the head, neck, or stalk of the polyp with no invasion of the base, and the carcinoma is well differentiated, no further resection is needed after simple snare polypectomy. The risk of residual cancer in the bowel wall or mesentery is very small, and recurrence or death due to cancer is very rare for such polyps.^{40,41} However, when the carcinoma has invaded the base of the polyp, is poorly differentiated, or shows lymphatic or vascular permeation, the risk of residual cancer in the bowel wall and adjacent lymph nodes is significant; formal surgical resection is indicated for these lesions.^{40,41}

Invasive carcinoma in a sessile polyp raises much more concern because the malignant cells—being so close to the bowel wall—may have already crossed the muscularis mucosae and invaded the submucosa. Complete endoscopic polypectomy of a sessile polyp, with microscopically clear margins free of carcinoma, is much more difficult to achieve for the same reason. Often, the sessile polyp is removed in piecemeal fashion, making it difficult if not impossible to determine whether the deep margin of resection is free of cancer. Sessile malignant polyps with favourable features probably behave just like their pedunculated counterparts, and do not necessarily require additional resection after complete removal by endoscopic polypectomy.⁴² The indications for formal bowel resection for a malignant sessile polyp are basically the same as for a pedunculated polyp, but valid ad-

ditional indications include suspected deep margin involvement by cancer after piecemeal removal, or extensive carcinoma invasion of over one third of the polyp.

Early adenocarcinoma of the colon

Both early and more advanced cancers of the colon receive standard surgical treatment. The affected segment of bowel is resected, such as by right or left hemicolectomy, with mesenteric lymph node dissection and end-to-end anastomosis of the bowel. The post-operative functional results and quality of life are excellent. The five-year survival rate after resection of a cancer invading only the mucosa and submucosa exceeds 90%.⁵ When tumour invades the muscularis propria the survival rate is 60% to 80%. However, the survival rate drops to below 40% when metastasis to lymph nodes has occurred.⁵

Early adenocarcinoma of the rectum

Cancer of the upper rectum is treated by an abdominal approach (anterior resection) to remove the upper rectum and sigmoid colon with the mesentery and mesenteric lymph nodes, and end-to-end anastomosis of the bowel. Although minor changes in bowel habit occur after the operation, patients adapt themselves well over time.

Cancer of the lower rectum, being close to the anus and pelvic floor, has traditionally been treated by wide lateral and distal dissection with complete rectal and anal extirpation and the creation of a permanent colostomy. While large and advanced rectal cancers may still need such radical treatment today, most early cancers of the low rectum can be adequately resected with preservation of the anal sphincter. Wide lateral dissection is similarly performed, but the anal sphincter may be preserved provided a resection margin of 1.5 cm to 2.0 cm below the rectal cancer is achieved.^{43,44} The quality of life is obviously better with preserved anal sphincter function. Five-year survival rates of 80% to 90% can be achieved for cancers limited to the mucosa and submucosa, and 70% to 80% if cancer invades the muscularis propria, provided there is no metastasis to the lymph nodes.⁴⁵

Clinical trials in North America showed that after sphincter-saving resection, the survival rate was similar to that after abdominoperineal resection, even though local recurrences were more common, particularly when the distal resection margin was less than 2 cm.⁴⁴ Anastomosis of the very limited anorectal stump to the colon requires precise technique, using either manual suturing or mechanical stapling. For very small low-rectal cancer, trans-anal removal by wedge bowel-

wall resection without formal bowel resection and anastomosis is another treatment option. The results are best if the tumour is well-differentiated and confined to the mucosa and submucosa of the lowest 5 cm of the rectum. Postoperative radiotherapy is usually given. Excellent functional results with a five-year survival rate of 90% and local recurrence rate of 8% can be attained.⁴⁶

In the past, many male patients have suffered impotence after resection of rectal cancer. This occurred when wide lateral surgical dissection on the pelvic side wall injured the hypogastric (sympathetic) nerves or the sacral parasympathetic nerves. Familiarity with the anatomic location of these nerves during rectal resection is important if iatrogenic impotence is to be avoided, but sacrifice of these nerves may be necessary in advanced cancers infiltrating the pelvic sidewall.⁴⁷ For early cancer of the rectum, lateral invasion is rare, and careful dissection should completely avoid such nerve injury.

Opportunities for early diagnosis and treatment in Hong Kong

According to the Annual Department of Health Report (1992-93) of Hong Kong, 2002 new cases of CRC were reported to the cancer registry, and 8364 hospital inpatients were treated for the disease.¹ While it is not known how many were early and how many were advanced cancers at the time of diagnosis, the fact that the number of treated inpatients was more than four times greater than the number of new cases would suggest that most of these cancers were advanced. The 8364 hospitalised patients represent considerable morbidity as well as economic cost. For a population of approximately six million, two million were over the age of 40 and thus at risk for this disease. The 8364 inpatients translate into 4 of 1000 persons in this age group being thus disabled, annually.

Primary prevention of CRC must await new knowledge as to its cause. Meanwhile, only early diagnosis will allow simple and effective treatment with high cure rates and low morbidity. The FOBT is a reasonable screening method for all persons over the age of 45. The test kits are relatively simple to use and are not unreasonably expensive. At least one non-profit cancer-detection organisation in Hong Kong offers the test to targeted civilian groups and by mail. A small number of private clinics are also offering the test to patients. However, given the low sensitivity of FOBT and the currently low penetration of the target population by FOBT, the yield of

diagnosed early colorectal cancer has been disappointing. Only public education can improve this. A substantial increase in the yield will only come if many thousands more ask for the test. Such a screening campaign should be considered by the government and the medical profession.

Clinical targeted screening and surveillance can be further pursued. Currently, patients found to have adenomas or those already being treated for CRC are being closely followed. Much greater vigilance is needed for the many thousands of individuals who suffer from supposed "haemorrhoids" or have one or more first degree relatives who have been treated for colorectal adenoma or cancer. Very few of these individuals are currently being told or counselled about their risk of developing CRC, and of the opportunity for early diagnosis and treatment. Cost is an obvious concern, and resources may not allow all of these individuals to undergo colonoscopy. But at least an FOBT, if not barium enema or flexible sigmoidoscopy, can be advised. Over time, the cost savings due to early diagnosis from such endeavours may well exceed the expense of caring for 8364 hospitalised patients annually.

References

1. Lee SH. *Department of Health (Hong Kong) Annual Report 1992/1993*. Hong Kong: Hong Kong Department of Health; 1993.
2. Fuchs CF, Giovannucci EL, Colditz GA, Hunter DJ, Speizer FE, Willet WC. A prospective study of family history and the risk of colorectal cancer. *N Engl J Med* 1994;331:1669-74.
3. Burt RW, Bishop DT, Cannon-Albright L, et al. Hereditary aspects of colorectal adenomas. *Cancer* 1992;70:1296-9.
4. Ekblom A, Helmick C, Zack M, Adami HO. Ulcerative colitis and colorectal cancer: a population based study. *N Engl J Med* 1990;323:1228-33.
5. Cohen AM, Minsky BD, Schilsky RL. Colon cancer. In: DeVita VT, Hellman S, Rosenberg SA, editors. *Cancer: principles and practice of oncology*. Philadelphia: JB Lippincott Company, 1993;929-77.
6. Hamilton SR. Colorectal cancer in Crohn's disease. *Gastroenterology*. 1989;89:398-407.
7. Correa P. Epidemiology of polyps and cancer. In: Morson BC, editor. *The pathogenesis of colorectal cancer*. Philadelphia: WB Saunders, 1978:126-52.
8. Vogelstein B, Fearon ER, Hamilton SR, et al. Genetic alterations during colorectal tumour development. *N Engl J Med* 1988;319:525-32.
9. Winawer SJ, Zauber AG, Ho MN, et al. Prevention of colorectal cancer by colonoscopic polypectomy. *N Engl J Med* 1993;329:1977-81.
10. Gloeckler-Ries LA, Hankey BF, Edwards BK, editors. *Cancer statistics review: 1973-1987*. Bethesda, Md: Department of Health and Human Services, 1990.
11. Chung EC, Lau PC, Wong JC. Clinico-pathological study of colorectal adenoma in Hong Kong Chinese patients. *HK Pract* 1993;15:2879-83.

12. Cheung PS, Wong SK, Boey J, Lai CK. Frank rectal bleeding: a prospective study of causes in patients over the age of 40. *Postgrad Med J* 1988;64:364-8.
13. Ahlquist DA. Occult blood screening: obstacles to effectiveness. *Cancer* 1992;70:1259-65.
14. Macrae FA, St John DJ. Relationship between patterns of bleeding and Hemoccult sensitivity in patients with colorectal cancers or adenomas. *Gastroenterology* 1982;82:891-8.
15. Bang KM, Tillet S, Hoar SK, Blair A, McDougall V. Sensitivity of faecal Hemoccult testing and flexible sigmoidoscopy by HemoQuant in the postoperative surveillance of colorectal neoplasia. *J Occup Med* 1986;28:709-13.
16. Flehinger BJ, Herbert E, Winawer SJ, Miller DG. Screening for colorectal cancer with faecal occult blood test and sigmoidoscopy: preliminary report of the colon project of Memorial Sloan-Kettering Cancer Center and PMI Strang Clinic. In: Chamberlain J, Miller AB, editors. *Screening for gastrointestinal cancer*. Toronto: Hans Huber Publishers, 1988:9-16.
17. Kronborg O, Fenger C, Sondergaard O, Peddersen KM, Olsen J. Initial mass screening for colorectal cancer with faecal occult blood test. *Scand J Gastroenterol* 1987;22:677-86.
18. Gilbertsen VA, McHugh R, Schuman L, William SE. The early detection of colorectal cancers: a preliminary report of the results of the occult blood study. *Cancer* 1980;45:2889-901.
19. Hardcastle JD, Thomas WM, Chamberlain J, et al. Randomized controlled trial of faecal occult blood screening for colorectal cancer: the results of the first 107,349 subjects. *Lancet* 1989;1:1160-4.
20. Kewenter J, Bjork S, Haglind E, et al. Screening and rescreening for colorectal cancer: a controlled trial of faecal occult blood testing in 27,700 subjects. *Cancer* 1988;62:645-51.
21. Mandel JS, Bond JH, Church TR, et al. Reducing mortality from colorectal cancer by screening for faecal occult blood. *N Engl J Med* 1993;328:1365-71.
22. Fork FT, Lindstrom C, Ekelund GR. Reliability of routine double-contrast examination (DCE) of the large bowel in polyp detection: a prospective study. *Gastrointestinal Radiol* 1972;8:163-72.
23. Gelfand DW, Ott DJ. Single- versus double-contrast gastrointestinal studies: critical analysis of reported statistics. *Am J Roentgenol* 1980;137:523-7.
24. Dodd GD. The role of the barium enema in the detection of colonic neoplasms. *Cancer* 1992;70:1272-75.
25. Bolin S, Franzen L, Nilsson E, Sjobahl R. Carcinoma of the colon and rectum missed by radiologic examination in 61 patients. *Cancer* 1988;61:1999-2008.
26. Gilbertsen VA. Proctosigmoidoscopy and polypectomy in reducing the incidence of rectal cancer. *Cancer* 1974;34:936-9.
27. Selby JV, Friedman GD, Quesenberry CP Jr, Weiss NS. A case-control study of screening sigmoidoscopy and mortality from colorectal cancer. *N Engl J Med* 1992;326:653-7.
28. Newcombe PA, Norfleet RG, Storer BE, Surawicz TS, Marcus PM. Screening sigmoidoscopy and colorectal cancer mortality. *J Natl Cancer Inst* 1992;84:1572-5.
29. Maule WF. Screening for colorectal cancer by nurse endoscopists. *N Engl J Med* 1994;330:183-7.
30. Morson BC. Evolution of cancer of the colon and rectum. *Cancer* 1974;34:845-50.
31. Winawer SJ, Zauber AG, Diaz B. Temporal sequence of evolving colorectal cancer from the normal colon. *Gastrointest Endosc* 1987;33:167-73.
32. Lee T, Barringer M, Myers RT, et al. Multiple primary carcinomas of the colon and associated extracolonic primary malignant tumours. *Ann Surg* 1982;195:501-5.
33. Mecklin JP. Frequency of hereditary colorectal carcinoma. *Gastroenterology* 1986;90:328-33.
34. Mecklin JP, Jarvinen HJ. Clinical features of colorectal carcinoma in cancer family syndrome. *Dis Colon Rectum* 1986;29:160-4.
35. Hixon L, Fennerty M, Sampliner R. Tandem colonoscopy to define the polyp-miss rate of colonoscopy. *Gastrointest Endosc* 1990;36:210-4.
36. O'Brien M, Winawer SJ, Waye JD. Colorectal polyps. In: Winawer SJ, Kurtz RC, editors. *Gastrointestinal cancer*. New York: Gower Medical Publishers, 1992:3-30.
37. Riddell RH, Goldman H, Ransohoff DF, et al. Dysplasia in inflammatory bowel disease: standardized classification with provisional clinical application. *Hum Pathol* 1983;14:931-68.
38. Brostrom O, Lafberg R, Osta V, Reichard H. Cancer surveillance of patients with long-standing ulcerative colitis: a clinical, endoscopic and histologic study. *Gut* 1986;27:1408-13.
39. Blackstone MO, Riddell RH, Rogers BH, Levin B. Dysplasia-associated lesion or mass (DALM) detected by colonoscopy in long-standing ulcerative colitis: an indication for colectomy. *Gastroenterology* 1981;80:366-74.
40. Pollard CW, Nivatvongs S, Rojanasakul A, Reiman HM, Dozois RR. The fate of patients following polypectomy alone for polyps containing invasive cancer. *Dis Colon Rectum* 1991;35:933-7.
41. Kyzer S, Begin LR, Gordon PH, Mitmaker B. The care of patients with colorectal polyps that contain invasive adenocarcinoma: endoscopic polypectomy or colectomy? *Cancer* 1992;70:2044-50.
42. Ehrinpreis MN, Kinzie JL, Jaszewski R, Peleman RL. Management of the malignant polyp. *Gastroenterol Clin North Am* 1988;17:837-50.
43. Williams NS. The rationale of preservation of the anal sphincter in patients with low rectal cancer. *Br J Surg* 1984;71:575-81.
44. Wolmark N, Fisher B. An analysis of survival and treatment failure following abdominoperineal and sphincter-saving resection in Dukes B and C rectal cancer: a report of the NSABP clinical trials. *Ann Surg* 1986;204:480-9.
45. Cohen AM, Minsky BD, Friedman MA. Rectal Cancer. In: DeVita VT, Hellman S, Rosenberg SA, editors. *Cancer: Principles and practice of oncology*. Philadelphia: JB Lippincott Company, 1993:978-1005.
46. Bailey HR, Huval WV, Max E, Smith KW, Butts DR, Zamora LF. Local excision of carcinoma of the rectum for cure. *Surgery* 1992;111:555-61.
47. Enker WE. Potency, cure and local control in the operative treatment of rectal cancer. *Arch Surg* 1992;127:1396-402.
48. Winawer SJ, Enker WE, Levin B. Colorectal cancer. In: Winawer SJ, editor. *Gastrointestinal diseases*. Vol 2(27). New York: Gower Medical Publishing, 1992:1-39.