1963

Adenomatous polyps of the large intestine and their relationship to malignancy

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ADENOMATOUS POLYPS OF THE LARGE INTESTINE AND THEIR RELATIONSHIP TO MALIGNANCY

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Submitted in Partial fulfillment for the Degree of Doctor of Medicine

College of Medicine, University of Nebraska

April 1, 1963

Omaha, Nebraska

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INTRODUCTION

Although the true incidence of adenomatous polyps of the colon and rectum is unknown, data from cancer-detection centers estimate 2 to 7 per cent of the population have undetected polyps[1,2] estimates run as high as fifty per cent of individuals over age thirty. If the entire colon is examined, polyps will be found in ten per cent of the colons at necropsy.[4]

The exact nature of this lesion, which effects such a large segment of the population, is unknown; and what is considered adequate therapy is controversial. This paper will examine the theories concerning adenomatous polyps in the adult colon and their relationship to malignancy. Familial polyposis, villous adenomas, polyps secondary to chronic inflammatory processes, lymphomas, and other uncommon lesions which may present as a polypoid mass in the colon are excluded from this study.

HISTORICAL BACKGROUND

One of the earliest references to polyps in modern medical literature was the description by Menzel in 1721. He described a colon taken from a soldier dying of chronic dysentery. In it was a general inflammation of the intestinal tract and within the colon a number of wart-like elevations.[5] The next reference to polyps occurred in 1832 when Wagner described the methods of healing of dysenteric ulcers.

He noted tiny polypoid excresences on the margins of scars and on the surface of the cicatrix of the healed ulcers. Luschka in 1861 described a colon with thousands of polyps from the ileocecal valve to the rectum. He added a microscopic description to the literature. He wrote that the glands resemble the glands of Lieberkuhn, except they were longer and tended to be more branched. Some of the glands were dilated with crypt-like spaces. There was no change in the mucus membrane between glands.[7]

Woodward was the first to differentiate primary(true) polyps from those secondary to chronic ulcerative colitis (pseudo-polyps) in 1881. A year later Crypps described multiple polyposis of the colon associated with hereditary factors.[8]

PATHOGENESIS

The causitive factor or factors in the pathogenesis of the adenoma which have been suggested are inflammation, ruptured submucosal lymphoid tissue, and some inherent, localized defect in epithelial hyperplasia. During the 1920's the lymphoid theory was discussed. In essence this hypothesis developed from the observation that the lymphoid follicles which occupy portions of the mucosa and submucosa, breaking through the muscularis mucosa, will rupture into the lumen of the intestine forming ulcers.

The adjacent epithelium was thought to prolapse into the ulcers created. During the healing process some of the glands became entrapped and polyps resulted (the Schultze pattern).

Altwater reinvestigated this theory. He studied the discrete or conglomerate follicles in the colon. In his series 90 per cent of the pure controls (colons without evidence of polypoid formation) had evidence of ruptured follicles, 70 per cent of the colons with benign polyps, 72 per cent with sessile polyps and 82 per cent of the pedunculated polyps.[8] These statistics do not substantiate the lymphoid theory.

Saint believes chronic inflammation is the initiating factor in polypoid development. The basis of the premise is the presence of small round celled infiltrations and other cells of this type, indicative of chronic inflammatory process in the microscopic examination of the polyps and the development of the adenomas in thos parts of the bowel most liable to irritation.

The earliest recognizable microscopic change is in the epithelium of the normal glands. The adenoma evolves through hypertrophy and hyperplasia of glandular tissue, creating a slight thickening of the mucosa which forms a small fold. An upward projection of the submucosa in the first suggestion of a pedicle.

He states pedicle development is due to drag upon the adenoma by the constant passage of feces over it and the peristaltic contractions of the bowel attempting to expel the developing adenoma which the bowel considers a foreign body.

The adenoma increases in size through glandular proliferation. This is thought to be accomplished by the formation of new glands at the tip of the adenoma because the glands at the tip appear to be less differentiated and thus must be younger than those near the pedicle. The gland themselves often seem to be segmenting, thereby forming younger glands.[5]

Others agree that epithelial hyperplasia is the earliest microscopic feature of a developing adenoma but state no theories as to the etiology.[8,10,13] There is disagreement concerning the features of the developing adenoma.

Altwater, recognizes a site of rapid proliferation of epithelium within normal mucosa. He suggests the proliferation results from the removal of some unknown inhibitory power over normal proliferation, allowing overgrowth. This early glandular hyperplasia of epithelium has normal microscopic appearance and physiochemical function based on mucus production. The numerical increase in cells brings about an increase in the size of the crypts of Luberkuhn with elongation of glands which is limited by the muscularis mucosa. The limitation of

downward growth forces the increasing glands to project into the lumen of the bowel, causing the elevation on the mucosal surface. With overgrowth comes tubular branching.

The cells multiply faster than a given segment of structure can accomodate. With this rapid multiplication the nuclei are piled one on top of the other, losing their normal position near the basement membrane, and moving closer to the lumen of the tubule. There is an increase in the amount of cytoplasm that is between the base of each cell and the nucleus.

With the loss of cellular allignment, the cells become cuboidal in shape. There is decrease in mucus production. Nuclear outline changes from loose fusiform shape to spindle shape. The chromatin content increases and becomes vesicular in character. Mitotic figures are found in greater number.[8]

The microscopic description of Bacon and his coworkers is a uniform proliferation of glands, regular in size and shape. The lining cells are usually of the tall columnar variety with frequent goblet cells, and one row deep. In the earliest form of developing adenoma the cells are regularly arranged with no intra-glandular budding or stratification. Nuclei show little if any differences in size or staining properties; mitosis are

absent or infrequent. They found it difficult to discern between the young adenoma and focal hyperplasia.

When the adenoma becomes pea to cherry sized, Bacon's microscopic description is the same as Saint's findings. With growth of the adenoma come branching and cup like enlargement of the glands.

There is beginning loss of cellular order in relation to the connective tissue stalk but with intact basement membrane. Larger adenomas have a central stalk with secondary branches, (fronds) that support the polypoid processes. It is composed of connective tissue and smooth muscle. The stroma may be edematous, cellular or composed of dense hyalinized material. Large stalks contain arteries and veins that may be thickened, hyalinized or dilated and hyperemic. Large amounts of hemosiderin or extravascular erythrocytes are sometimes found.[11]

Helvig describes elongation, dilatation, branching, and sometimes, papillary infolding of the glands. The columnar cells are only slightly different from normal. The columnar cells may be elongated or compressed, with or without mucus. The nuclei are uniform, have moderate amount of chromatic material, and usually have no large nucleoli. He does not find marked stratification of cells and the nuclei occupy the basal zone.

A distinct demarcation is found between cells and the adjoining stroma in his series.

According to Duke's work, the developing adenoma arises from small areas of increased epithelial growth which is characterized by nuclei that stain more deeply with H and E stain and by decreased mucus production. Due to the active growth of the epithelium, there is a projection into the bowel lumen. This is accompanied by a bowing of the muscularis mucosa. There is an elongation of the connective tissue and vascular element of the muscularis mucosa. The bending of this layer of bowel wall provides a more extensive base to support the new growth. There is a continuous lengthening and branching of the scaffolding structures with growth of the adenoma.[12]

The initial feature described by Valdes-Dopnea is not epithelial hyperplasia but swelling of the submucosa, causing upward displacement of the muscularis mucosa. This feature gives the small, raised, dome-shaped projection on the crest or folds and not epithelial hyperplasia. This may be present without evidence of epithelial hyperplasia.

He believes the adenoma never begans as pure epithelial proliferation. There maybe a primary, subtle

change in the epithelium that is not histologically demonstrable. This non-visualized factor may produce a reaction within the submucosa that is transmitted into a nodular swelling. The epithelial change may then progress into visual hyperplasia. In some cases these changes may become evident before formation of the pedicle, in others there is a mucosal projection first. The swelling creates the pedicle.[14]

Most writers on pathogenesis of adenomas agree that one of the earliest microscopically detectable findings is epithelial hyperplasia. The initiating factor and steps in development are points of disagreement. No controlled studies of the genesis of an adenoma have been done and no polyp in the human has been followed to full development with histologic evidence of the transitional phases. Therefore, pathogenesis of the adenoma will remain conjecture until controlled studies have been made.

STATISTICS

Statistics, the basis for arguments on the adenomatous polyp's supposed relationship to malignancy, are derived from three sources of material, autopsy, surgical specimens and colon and rectal examinations of symptomatic and asymptomatic patients. The most extensive necropsy study to date was done by Helvig.

This series consisted of 1460 consecutive autopsies. The entire colon was examined with a hand lens for polyps. Serial sections were prepared from all polyps found. The following table is a composite of some of his findings relating to incidence of polyps.

TABLE I Incidence of Polyps in Necropsy Specimens

Age (yrs) Group	No. of Pt's.	No. With Polyps	& With Polyps	Single Polyps	Multiple Polyps
0-1	284	0	0	0	0
1-10	85	3	3.4	2	1
11-20	70	2	2.5	2	0
20-30	96	0	0	0	0
31-40	132	9	7.5	5	4
41-50	161	11	6.2	6	5
51-60	255	337	14.5	21	16
61-70	206	42	20.3	22	20
71-80	134	331	23.1	18	13
81-90	25	4	16.0	11	3
91-100	2	0	0	0	0
Totals	1460	139	9.5	77	62

TABLE II Age and Incidence of Carcinoma in White Patients

Age (yrs) Group	No. of Cases	No. of Polyps	Presence of Carcinoma in Polyps		in Mucosa			
			No.	%	No.	%	No.	%
0-1	228	0						
1-10	79	2						
11-20	73	1						
21-30	182	1	1	1.5				
31-40	110	9	2	1.8	1	0.9		
41-50	138	11	3	2.2	2	1.4		
51-70	188	40	11	5.8	6	3.2	1	0.5
71-80	124	30	3	2.4	1	0.8	1	0.8
81-90	24	4						
91-100	1							

These figures imply there is a progressive incidence of polyps, peaking in the seventh decade and

a corresponding increase in the incidence of multiple polyps with age. The author does not state whether he included the histologically atypical polyps in the groupings of carcinoma within a polyp or his criteria for malignancy. His reported malignancies peak at the sixth decade of life. The mean decade for both malignant and benign polyps is in the fifth decade.[8]

Adren and Frieberg examined 3609 symptomatic patients using the double contrast radiologic technique. The symptoms in the children was usually rectal bleeding. Adult patients' symptoms were not necessarily related to the colon, but colon studies were done were incidentally. The next table present their findings as to incidence of polyps.[15]

TABLE III

Incidence of Polyps Among Patients with Symptoms Related to Diseases of the Bowel

Age Groups	No. of Cases	No. with Polyps	% with Polyps
0-9	3	15	28
10-19	106	3	3
20-29	259	12	4.6
30-39	538	31	7.6
40-49	775	74	9.6
50-59	836	82	9.8
60-69	691	88	12.8
70-79	327	38	11.6
80-89	28	5	16.7
Totals	3609	347	9.6%

In a series of 246 surgical specimens, Backus et al had a mean age for their series of 55 years, a 12 per cent incidence for polyps and a slight predominence of males, 54 per cent. The distribution

of benign polyps and carcinomas of the colon is presented in Table IV. The figures are similar to those presented by other authors.[10,15,17,18]

TABLE IV
Distribution of Polyps and Carcinoma in the Large Intestine

Location	% of Polyps	% of Cancers
Rectum	41	34
Sigmoid	40	41
Descending Colon	9	3
Splenic Flexure	1.2	1.5
Transverse Colon	4	5.5
Hepatic Flexure	1.2	2.0
Ascending Colon	2.0	2.0
Cecum	0.5	6.0

THEORY I POLYPS ARE PREMALIGNANT LESIONS

The confusion surroundint the colorectal adenoma is due in part to the variable biologic pattern which they exhibit and the failure of many writers to state their criteria for diagnosis or malignancy.[19] Points generally accepted in the definition of colorectal adenoma are ; (1) pedunculated lesion; (2) glandular origin; and (3) projects into the bowel lumen.[4,13,19,20] Terms such as"benign" and "localized" are added by some authors, signifying their philosophy as to the ultimate fate of the lesion.

Malignant adenoma, adenocarcinoma Grade I, atypical adenoma, carcinoma <u>in situ</u>, and invasive adenoma are terms frequently found in the literature without precise definition by the authors using them. A specific adenoma should be thought of as either benign or malignant, the diagnosis being unequivocal.[13]

Evidence which suggests a premalignant nature for the adenomatous polyp is : (1) There is a similar distribution of adenomatous polyps and frank carcinoma. (2) They have similar age and sex incidence. (3) In almost one-third of surgical specimens removed for carcinoma of the large intestine, polyps are found in association with the cancer, and (4) Sometimes evidence suggestive of an adenomatous polyp will be found

at the edge of a malignant growth.[21]

Swinton and Shields, who believe that malignant change may begin anywhere in a polyp, established a Criteria for malignancy in 1939 which has been accepted by other authors, and in turn altered to suit the specific investigator's opinion.[2,11,22,23] The original criteria consisted of anaplasia, irregularity of architecture and invasion. For a diagnosis of malignancy, two of the above features must be present or invasion of lymphatics and blood vessels.[4]

Helvig expanded the criteria for malignancy stated by Swinton and Shields. He described anaplasia as a variation in size of cells and nuclei, deep staining nuclei with prominent nucleoli, and the presence of mitotic figures. Irregularity of architecture is manifested by excessive stratification of cells, intraglandular proliferation of cells not producing gland-like structures and cell masses which are not separated by a limiting membrane. Invasion is present when no definite border can be recognized between the epithelial cells and surrounding stroma.[10]

Multiple or serial sections should be taken to include the base of each polyp and the adjoining wall[10,24] because "many adenomas exhibit minor variations in structure in different parts, rather than a homogenous

picture...a few adenomas contained a focus or foci thought to represent the transitional stage between the benign and the malignant polyp.[10]

Morrison reserved the term malignant polyps for those adenomatous polyps in which there is unequivocally invasive carcinoma. He does not believe in the pre-invasive carcinoma, (carcinoma _in situ_) because a high proportion of the adenomatous polyps if examined by serial section, show much structural irregularity and epithelial hyperplasia. This is called by some authors carcinoma _in situ_. He feels the presence of invasion is necessary before an adenoma should be labeled malignant for then it has unmistakenly passed from the benign stage to become a malignant growth.[21]

Some authors have browken down the premalignant classification into "pre-malignant changes."[11,20,23] Starr describes polypoid epithelial hyperplasia--multicentric or unicentric -- as the earliest point of transition from benign to malignant polyp. The next progression to the malignant states he calls adenomatous polyps with atypical glands in the various proportions but not sufficiently atypical for carcinoma _in situ_.[21] A similar term is used by Bacon. He described adenoma with atypism as lesions which for the most part have one or more small foci of atypical

epithelial proliferation which "seems to be more than that seen in response to inflammation or degeneration and yet its exact status is difficult to assess." Small localized areas have varying degrees of hyperchromatism of nuclei, pleomorphism, minimal pseudostratification, beginning loss of polarity of the nuclei, occasional atypical mitotic figures.[10]

Starr proceeds in his classification of the "pre-precancerous polyp," describing adenomatous polyps with atypical glands and questionable invasion that could be the result of entrapped glands in the stalk or artifact. His next classification is benign glands not entirely free of signs of malignancy. Definite malignancy is present in this classification when there is polypoid infiltrating carcinoma with no benign glands.[20]

Bacon's definition of atypism a synonym for adenocarcinoma <u>in situ</u> overlaps Starr's multiple gradation of progression to frank malignancy. In defining this term, he states the character of the epithelial cells must be considered and the lesion should not be called benign or premalignant because it is within the confines of the basement membrane. Cytologic features should be a strong factor in correct labeling of a lesion. Such features as irregularity of gland pattern, pleomorphism, hyperchromatism, unbalanced mitotic activity of the component cells, loss of polarity, pseudostratification and secondary daughter acinar proliferation should be

the deciding features.[11]

Another author uses the term "carcinomatous change" for the "pre-premalignant" lesion. He describes the polyp as being clinically benign with foci of cells exhibiting the characteristics of carcinoma. He uses the term "carcinoma in situ" for the next progression towards malignancy, but does not define it. Invasive carcinoma is divided into three groups; (1) penetration of the mucosa muscularis; (2) partial penetration of muscularis mucosa and (3) invasion superficial to the muscularis mucosae.[23]

Other terminology for the lesions in the group that are definitely malignant varies among authors. In contrast to the above grouping of malignancies, Bacon prefers the term "adenocarcinoma Grade I". This stage of malignancy has the cellular characteristics of a malignant adenoma plus invasion of the basement membrane. Adenocarcinoma Grade II is defined as adenocarcinoma definitely arising from an adenoma.[10]

In surgical specimens of colons with frank carcinomas, 11 to 20 per cent have one or more grossly benign polyps,[2,11,23] and up to fifty per cent of carcinomas found at autopsy have associated benign polyps.[23] An example of such case studies is the following table compiled from Bacon's series. In his series of 800 cases, 162 cases had adenomatous polyps in the surgical specimen, or 20 per cent.

Other authors have similar findings if regrouping their cases is done in order to have the same criteria for each group. His cases were placed in three groups, benign polyps -- no microscopic evidence of atypism, premalignant polyps -- all stages of atypism, including carcinoma in situ, and invasive carcinoma.

TABLE V
Incidence of Polyps associated with Carcinoma of the Colon in Surgical Specimens

Groups	Number of Polyps	% of all Polyps
Benign Polyps	26	16
Associated with CA	4	8.6
Premalignant Polyps	77	47.5
Associated with CA	31	18.9
Invasive CA within a polyp	59	36.4
Grade I	24	14.8
Associated with CA	3	1.8
Grade II	5	3.0
Presumptive evidence of Adenomatous origin	30	18.7

It has been suggested that "the polyp is a signal, indicating abnormal mucosa prone to develop either new polyps with varying degrees of malignancy or carcinoma. In a series of 537 patients with a history of polyps, followed for a ten year period, there averaged a ten per cent per year recurrence, with no particular period of greater or lesser recurrence rate.[2]

Realizing the difficulties in distinguishing between artifact in histological preparations, atypism, and reaction to inflammation in the adenomatous polyp, workers who believe that adenoma is a precursor to carcinoma

feel they can demonstrate progression from the benign to malignant states in any large series of polyp studies.[20,23,24] They think the adenomatous polyp in a significant, but unknown number of cases, result in cancer of the colon. In their experience there has never been a case of metastatic disease from a polyp showing premalignant changes without invasion. Carcinoma may arise in areas of colonic mucosa other than in a polyp. Because the course of given polyp cannot be predicted, all colorectal adenomas should be removed in absence of systemic contraindication as a matter of cancer prophylaxis even if only a small percentage eventually become malignant.[19]

THEORY II ADENOMATOUS POLYPS ARE NOT PREMALIGNANT LESIONS BUT ARE BENIGN OR MALIGNANT FROM THE BEGINNING OF THEIR DEVELOPMENT AND DO NOT CHANGE OVER THE COURSE OF TIME

All variations from frank carcinoma to minor degrees of atypia within the polyp can be found in any large groups of polyps.[25] It has been estimated 10 to 15 per cent of polyps have one or more areas of atypical changes sufficiently definite to be considered intramucosal carcinoma. Smaller percentages show definite invasive carcinoma arising somewhere within the polyp. The exact incidence of carcinomatous changes varies with the personal opinion of the pathologist as to the degree of atypica necessary for him to consider a specific lesion malignant.

Koppel et al.,examined 4,400 patients over 36 years of age without previous history of polyps of colon or rectum, ulcerative colitis, or any major bowel operations. The minimum bowel examination procedure was the use of a ten inch Sigmoidoscope. The majority of the patients had no symptoms of organic disease of the colon or rectum. Of the 4,400 patients studied, 19 carcinomas without polyps, 9 carcinomas associated with polyps developed during a five-year follow-up. There were 307 patients with polyps for a prevalent ratio of 7 per-cent.* They found among patients free of polyps at the time of

*Prevalent ratio is the proportion of general population having a stated characteristic at any specific time.

sigmoidoscopy, the subsequent incidence of cancer of the colon and rectum was almost as high as the incidence in the general population. The ratio for the patient with adenomatous polyps is even lower than those without polyps. In sample studies there was neither high cancer incidence rate *among patients with treated polyps nor a significantly low rate among polyp free patients. Patients with benign adenomatous polyps may have an increased risk of developing cancer but their studies indicates this risk must be relatively small during the first 5 years after the discovery of the first polyp. Table VI is a summary of the work done by Koppel et al.

TABLE VI

Incidence of New Cancers During a Five-Year Follow-Up of Patients who were initially Asymptomatic

Group	No. of Pts.	New Cancers in 5 years		Ratio
		Observed	Expected	
Total Studied	4,400			
No. Polyps	4,093	19	22.04	86
Benign Polyps	307	1	2.06	49

The studies of Colvert and Brown, as shown in Table VII support the findings of Koppel et al.

TABLE VII

Five-Year Follow-Up of Patients with Rectal Polyps

Groups	Number	Per Cent
Total Number of polyps removed at first examination	167	
Carcinoma present on first examination	10	5.9
Benign polyps removed on first examination	157	
Cancers found within 5 yrs. of first exam.	3	2.5
Total carcinomas in 5-yrs.	13	8.4
Polyps not removed on first exam.	43	
Carcinomas found when polyps removed 5-years later	3	6.9

*Incidence rate is the rate of occurrence of the characteristic being studied during a specific time.

These figures indicate there is not a significant difference in incidence of malignancy in patients having had their polyps removed and those which were followed for five years. If benign rectal polyps become malignant in time, one would expect a much higher incidence of malignancies in polyps that have been present for five or more years than in those patients who had their polyps removed. The conclusion drawn is that malignant polyps are either malignant in the beginning or become so very early; benign polyps do not become malignant with passing of time.[30]

Spratt, et al have noted polyps associated with single cancers within the rectum and sigmoid colon are located proximal to their respective cancers; polyps are located distally to carcinomas in the right colon. If cancers originate in adenomatous polyps then two adenomatous polyps arising simultaneously, both have equal chances of becoming the cancer and, consequently, the one that does not become cancerous. Therefore, there should be an equal incidence of the associated polyp being either distal or proximal to the cancer, regardless of its location. The co-existence of polyps with a frank carcinoma does not alter the distribution of the cancer within the colon, which would be expected

if a sufficient number of adenomas become cancers.[31]

Regardless of histology, only those polyps which show invasion of the stalk exhibit clinical characteristics of carcinoma. Epithelial atypical should not be diagnosed as carcinoma and should always be treated conservatively. "The overwhelming majority of cancers of the colon arise as cancer de nova..." The adenomatous polyp is a lesion of negligible malignant potential.[32]

DISCUSSION

Those who believe the polyp is a premalignant lesion, feel that any polyp, at any time may began to exhibit carcinomatous changes and will progress to frank carcinoma if permitted to remain in the colon. Opponents of the view believe the adenomatous polyp shows carcinomatous change very early in its development if it is to be a malignant lesion.

Both sides recognize the presence of foci of anaplasia and glandular irregularity within a polyp. Disagreement is exhibited over the significance of these particular characteristics. Polyps with these two characteristics do not have the other features of carcinoma, local extension, metastases, or local recurrence. Invasion of the stalk by atypical cells is necessary before the polyp develops the characteristics of clinical malignancy.[11,16,23,32]

Invasive adenocarcinoma may present as a polyp. Only histologic examination of the adenoma can determine if a malignancy exists. Therefore all polyps should be removed at their base for histologic study.[33]

Clinically, the arguments have little significance at the present time. All polyps must be biopsied to determine if actual malignancy is present because no other means for diagnosis is available. Because metastases only occurs after invasion of the stalk, total excision and fulguration of the base is adequate therapy for polyps exhibiting atypism without invasion.[14,16,32,33]

SUMMARY

The adenomatous polyp was mentioned early in modern medical literature but it was not until 1881 that true adenomatous polyps were differentiated from pseudopolyps of chronic inflammatory disease. Epithelial hyperplasia and glandular hypertrophy are the two earliest characteristics in the pathogenesis of the polyp. Possible etiological factors suggested are inflammation, ruptured submucosal lymphoid tissue and some inherent localized defect in epithelial hyperplasia. There is no proof for the validity of any of these theories.

Some investigators believe that in time, a significant number of adenomatous polyps will develop malignant characteristics.

Others think the adenomatous polyp is either benign or malignant at its beginning or very early in its development. The review of the literature failed to produce sufficient evidence to support one view above the other. Both groups agreed a malignancy may be present in the form of a polyp without the gross appearance of malignancy.

To determine if malignancy is present, biopsy of the total polyp, including the base is necessary. Therefore, all polyps should be removed for histologic study unless there are systemic contraindications. Because metastases do not occur unless there is invasion of the stalk, adequate therapy for polyps exhibiting atypical epithelial changes but not invasion of the stalk, is excision and fulguration of the base.

CONCLUSION

1. There is not sufficient evidence to support the theory of the premalignant nature of the adenomatous polyp, or the converse of the statement.
2. Polyps exhibiting characteristics described as "pre-malignant" can be adequately treated by local excision with fulguration of the base because metastasis does not occur unless invasion of the stalk is present.

ACKNOWLEDGEMENT

I wish to thank Dr. John Brush for his enthusiasm, cooperation and knowledgeable assistance in the preparation of this paper.

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