cular sites with biopsy proof. Until colonoscopy television and videotape records become practical, the colonoscopic diagnosis rests purely with the investigator and depends on his expertise only. Colonoscopy is also limited by technical difficulty at strictures and diverticular disease whereas rarely does the enema fail to demonstrate these difficult segments.

We believe that radiologists and endoscopists should therefore work as a diagnostic team, the barium enema remaining the routine method of investigation and colonoscopy being used for examination of detailed problem areas enabling pathological proof to be obtained. We feel that one of the earliest benefits of endoscopy may be an improvement in the standard of routine radiological examination of the large bowel.

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Epidermoid Cysts and Polyposis Coli

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Gardner's syndrome is usually described as a triad of soft tissue tumours, hard tissue tumours and polyposis coli (Gardner & Richards 1953). Many other features have been described since, of which the most important are desmoid tumours (O'Brien & Wels 1955), abnormalities of the teeth (Gardner 1962, Fader *et al.* 1962), duodenal polyps and carcinoma of the ampulla of Vater or duodenum (Melmed & Bouchier 1972).

The most commonly recorded soft-tissue tumours are sebaceous cysts which have been described as sebaceous cysts, epidermoid inclusion cysts, sebocystomatosis (steatocystoma multiplex) or atheromas. These terms appear to have been used interchangeably as if they all represent the same condition. However, 'sebaceous cysts' are not a single entity but any one of three conditions, that is, pilar cysts, epidermoid cysts or steatocystoma multiplex. These three conditions have different clinical features and are histologically quite distinct.

Present Study

Fifteen families with polyposis coli were examined in detail to determine which type of cyst occurs in Gardner's syndrome and details of 108 possibly affected individuals were recorded. Of these, 74 had polyposis coli; 5 died of carcinoma of the colon or rectum under the age of 40 and were probably affected; 5, aged between 20 and 30 showed no polyps on sigmoidoscopy and were probably unaffected; 24 under 17 either showed no polyps on sigmoidoscopy or were too young to have been sigmoidoscoped. Of the 74 definitely affected patients, 39 (53%) also had cysts of the skin. All patients who had cysts also had, or later developed, polyps of the colon. Individual patients were found to have between 1 and 20 cysts, with an average of 4 per person. They were situated most commonly on the legs, face, scalp and arms; the trunk was relatively uncommonly affected.

The cysts developed at any time from birth to 35 years, with an average age of onset of 13. This was statistically significantly earlier than the colonic polyps and confirmed previous observations that the cysts might be used as a guide to indicate which members of a family would develop polyps (Gardner & Richards 1953, Coli *et al.* 1970, Rosten 1972).

In 3 of the 15 families studied all the affected members had cysts; in the other families approximately half had cysts. Therefore the occurrence of cysts is not a completely reliable means of detecting affected members of a family.

Histological examination of 21 cysts from 14 patients all showed the characteristic features of epidermoid cysts. Pilar cysts and steatocystoma multiplex were not seen. Epidermoid cysts are very uncommon in children and those who develop them before puberty should probably have a sigmoidoscopy every three years from the age of 14, whether or not there is a family history of polyposis coli. Individuals of any age who are found to have pilar cysts or steatocystoma multiplex do not need sigmoidoscopy.

The diagnosis of Gardner's syndrome is often difficult because many of the patients do not fit precisely into the definition given by Thomas *et al.* (1968). In this clinical study, 39 (53%) of the definitely affected patients had cysts, 18 (24%) had osteomas, 13 (18%) had abnormal teeth, 5 (7%) had desmoid tumours and 5 (7%) had malignant tumours other than of the colon and rectum. These changes were present in different combinations in different families and in different members of the same family.

In some families there were affected members who showed none of these extra features and had previously been considered to be suffering from familial polyposis and not Gardner's syndrome.

Conclusions

At the present time it is not possible to say whether Gardner's syndrome is the same as or different from familial polyposis. However, if all individuals with polyposis coli were to have a full clinical examination and X-rays of their skull, jaws and long bones, looking for cysts, osteomas and dental abnormalities, it should be possible to answer this question in due course.

The cysts that occur in Gardner's syndrome are not pilar cysts or steatocystoma multiplex but epidermoid cysts. In view of their rarity in children, any child who presents with them should be considered as possibly having Gardner's syndrome and examined at regular intervals by sigmoidoscopy from the age of 14.

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Diverticular Disease

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Diet and Diverticular Disease

In his Harveian Lecture, Sir Berkeley Moynihan (1927) stated 'A diet leaving little residue is the one generally advised but I am not sure that a diet leaving a bulky residue is not better, providing that the bowels act once per day.' Nevertheless, until a decade ago a low residue diet continued to be recommended in the medical management of the disorder. Following colonic motility studies by Painter & Truelove (1964), Arfwidsson (1964), and Parks & Connell (1969), attention was again focused on the possible relevance of diet to the pathogenesis of colonic diverticula.

It is now generally held that a low residue diet often results in slow transit of the intestinal content through the colon, thus allowing time for the absorption of more fluid from the residue, frequently resulting in constipation.

The rationale behind the use of a high residue diet is that the colon has to deal with a large volume of fæces and thus maintains a greater diameter. In such a colon, segmentation is less efficient and the walls cannot be brought into opposition so readily and intraluminal pressure tends to be lower.

Epidemiological Aspects

Painter & Burkitt (1971) have pointed out that the introduction of roller milling, about 1880, resulted in a diminution in cereal fibre in the flour and that this together with reduction in the amount of bread consumed by individuals has an important bearing on the etiology of diverticular disease of the colon. Cleave *et al.* (1969) have shown that the death rate from diverticular disease has increased progressively since the early 1920s except during the war and immediate post-war period when white bread and sugar were restricted. They felt that these facts are interconnected.

Epidemiological studies also confirm that the disease is not racial in origin. Painter & Burkitt (1971) pointed out that the African native scarcely ever suffers from the disease whereas it is common in the American negro. Attention has been drawn to the fact that the disease is common in those of Japanese origin living in Hawaii or