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CLASSIFICATION OF DCIS; CLINICAL CONSEQUENCES

J.L. Peterse
Department of Pathology, The Netherlands Cancer Institute, 1066 CX Amsterdam, The Netherlands

Treatment of the increasing number of screen detected non-invasive breast cancers is a major problem. The classical ablative, therapy results in nearly 100% cure rate, but may be an overtreatment. The alternative, breast conservation, is being studied in running randomized trials; answers to the most important end points, risk of dying of metastasized. recurrent disease, or of side effects of treatment, requires long follow up.

In patients, not eligible for the trials, several tumour characteristics play a role in treatment choice. The *extent* defines the possibility of breast conservation. The *adequacy of excision* (min. tumour free margin) may be used to define the need for radiotherapy. As the *well differentiated DCIS* seem lesions with a low risk of transition into invasive carcinoma, and the subsequent carcinomas are of low grade malignancy, a wait and see policy with close, mammographic and clinical follow up may be applied for these lesions.

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DUCTAL CARCINOMA *IN SITU*—PROGNOSTIC SIGNIFICANCE OF HISTOLOGICAL TYPES

H.-E. Stegner
Department of Gynecology and Obstet. University Hamburg 20251 Hamburg, Germany

In a controlled clinical trial 114 patients with biopsy proven ductal carcinoma *in situ* were treated by breast conserving surgery without postoperative radiation. Intraductal comedocarcinoma was found in 41%, cribriform and papillary types were found in 22%. The proportion of high-grade lesions and low-grade lesions being 1:0.6.

Recurrences were found in 19%. High-grade DCIS was associated with a 15.3%, low-grade DCIS with a 12.2% recurrence rate. The recurrences did not show any correlation with the age of the patient nor the type and size of the lesion within the range of 2–46 mm. Optimal preconditions for mammographic follow-up seems to be of greater importance for the outcome of the patient than the size and type of the DCIS and the age of the patient.

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CONTROVERSIES IN DIAGNOSIS AND TREATMENT OF DUCTAL CARCINOMA *IN SITU* OF THE BREAST; THE SURGICAL ROLE

J.P. Julien, E.O.R.T.C. Breast Group
Centre H. Becquerel Rouen France

Mastectomy cures nearly 100% of DCIS and still now, wide-spread lesions need a mastectomy but the surgical role will include the possibility of an immediate or delayed reconstruction.

For small often infraclinic lesions a conservative surgery is advocated. This approach is difficult and may be hazardous if there is not a close collaboration between the radiologist, the surgeon and the pathologist. When an infraclinic lesion mainly microcalcifications have to be checked, the surgeon will decide if in order to locate the lesion he needs only two orthogonal views or will ask the radiologist to put a hook. He has to choose the best site of incision with a carcinologic but also cosmetic approach. The specimen is orientated by markers and the quality of the incision is immediately checked by X-ray. A wider excision may be immediately performed. The surgeon will not ask for a frozen section but will demand to the pathologist a complete detailed report with the nature of the lesions but also: the size, the location in the specimen, and a careful study of the margins. In our state of knowledge about DCIS the surgeon must always keep in mind that in a conservative procedure for a DCIS there is always a risk to miss the opportunity to cure the patient by a mastectomy.

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DOES RADIOTHERAPY INFLUENCE DCIS?

J. Harris
Harvard Medical School, Boston, U.S.A.

The influence of radiotherapy (RT) on DCIS can best be judged from randomized clinical trials. The situation is complicated by the heterogeneity of DCIS with regard to both pathology and extent so that subset analysis might be of importance. Also, since RT may only delay time to recurrence long FU is required.

Published results are only available from the NSABP trial. In the 1993 report, with a median FU of 43 months, breast recurrence was reduced from an annual rate of 5.1%/yr to 2.1%/yr with RT: RR = 0.40, $P < 0.001$. Of note in this trial is 1—the high rate of recurrence possibly due to the limited extent of surgery and degree of mammographic evaluation, 2—RR higher than the RR seen for invasive cancers (typically around 0.20), and 3—the apparent greater reduction in invasive compared with non-invasive recurrences. In the 1995 report, with a median FU of 48 months, the reduction with RT was similar for low-grade and high-grade lesions, but less for lesions without compared to with comedo-necrosis.

These short-term results indicate a positive influence by RT, but additional FU and the results of other trials will be necessary to make firm conclusions.

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PRIMARY PREVENTION OF LARGE BOWEL CANCER

J. Faivre, C. Couillault
Faculté de Médecine, 21033 Dijon Cedex, France

Epidemiological studies have emphasized the major role played by diet in the aetiology of large bowel cancer either as initiator, promoter or inhibitor of carcinogenesis. There is fairly consistent evidence concerning the protective effect of vegetables. There is some evidence relating fat intake, protein intake or caloric intake to colorectal cancer, while fiber intake or calcium intake seems to be inversely related to colorectal cancer. Available data are not sufficient to serve as a basis for firm, specific dietary advice. In the present situation, it is attractive to investigate available hypotheses within the framework of intervention studies. As there is considerable evidence that a high proportion of colorectal cancers arise in adenomas, adenoma recurrence and adenoma growth appear to be one of the most appropriate end points of intervention studies. Four intervention studies have been published so far. One of them suggests a protective effect of antioxidant vitamins on adenoma recurrence and three of them conclude in the absence of effect of these vitamins. A protective effect of lactulose on adenoma recurrence has also been suggested. Three studies are currently evaluating the effect of calcium supplementation, three studies the effect of fiber supplements, two studies the effect of antioxidant intervention. The results of these studies will be available within three years. If one of the evaluated interventions proves efficient, the benefits of a simple, safe and inexpensive prophylaxy for a very common cancer will be clear.

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IDENTIFYING RISK THROUGH FAMILY HISTORY ASSESSMENT

D.T. Bishop, N. Hall, B. Stephenson, P.J. Finan

Almost 40 years ago, Wolfe analyzed the family histories of a set of bowel cancer cases and showed that mortality through bowel cancer was over three times as common in the relatives of cases as in the relatives of controls. This basic observation has been repeated in a number of subsequent studies. More recent studies have attempted to delineate different family structures and the differences in risk between those structures. Two basic observations are apparent: (1) Relatives of early onset cases and (2) relatives of families with multiple cases are at particularly increased risk. For instance, relatives of cases diagnosed before the age of 45 years have a five-fold increased risk. Some of this increased risk must be due to the rare, dominant syndromes such as Hereditary Non-Polyposis Colorectal Cancer. While families which can be clearly diagnosed with this syndrome are rare, it may be that a considerable fraction goes undiagnosed. Alternatively, other genetic mechanisms or common exposures to environmental factors may be relevant. In summary, as a screening device, family history provides a valuable tool for recognizing a subset of those predisposed to bowel cancer.

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MOLECULAR DIAGNOSIS AND SURGICAL THERAPY OF HEREDITARY COLORECTAL CANCER

H.K. Schackert, M. Kadmon, C. Herfarth
Department of Surgery, Ruprecht-Karls-Universität D-69120 Heidelberg, Germany

Familial adenomatous polyposis (FAP) which is caused by APC gene mutations is a paradigm for presymptomatic molecular diagnostics and preventive surgical treatment. Sigmoidoscopy identifies symptomatic probands and presymptomatic relatives presenting with hundreds of colorectal adenomas. If sigmoidoscopy does not confirm FAP in relatives,