was observed among women born between 1870 and 1899. a total of 2,343 borderline turnouts were diagnosed between 1970 and 1993. The age-adjusted incidence rate has increased since 1970, reading 4.8 per 1000 000 person-years in 1989–93. The prognosis of ovarian cancer is still poor, and the crude 5-year relative survival was 36% in the Nordic countries in the late 1980s. in the present study, histology-specific long-term trench in prognosis of patients with ovarian cancer and borderline turnouts in Norway were examined The age-adjusted 5-year relative survival rate of patients with ovarian cancer increased steadily from 1954 to 1993. The increase in survival was most pronounced in women below the age of 65 years. No improvement was seen for women older than 75 years. The 5-year relative survival of the serous turnouts improved continuously from 1970 to 1993. For the mucinous tumours, an increase in relative survival was seen until 1984–88, thereafter the rate declined. In multivariate survival analysis, the RR of dying decreased with period diagnosis. For all patients with ovarian cancer, a RR of 0.5 (95% CI = 0.4–0.5) was seen in 1989–93 compared with 1954–58. Restricting the analysis to patients with epithelial cancer (197093), a RR of 0.6 (95% CI = 0.6–0.7) was seen in 1989–93 compared with 1970–73. In an analysis restricted to patients with epithelial cane., the patients with mutinous, endometrioid and clear cell turnouts had the lowest odds for having distant metastases. The age-adjusted 5-year relative survival rate of patients with borderline tumours was almost constant between 1970 and 1993, at a level of about 95% For these patients, age turned out to be the strongest prognostic factor. RRs of 11 and 34 were found for the age groups 65–74 and 75–89 years, respectively, compared with women younger than 45 years.

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Ovarian cancer: Progress in chemotherapy

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In the last decade it has been shown that long-term survival is achievable for women with advanced ovarian cancer. With cisplatin-based combinations 10 year survival rates are about 20 percent and even 15 years after treatment patients survive free of disease. The present standard of care is paclitaxel 135 mg/m^2 in 24 hours with cisplatin. It can be expected that with paclitaxel in initial treatment programs the long-term survival rate will increase. A shorter time of administration and an increase of the dose of paclitaxel will enhance the incidence of neurotoxicity. For this reason carboplatin (a less toxic cisplatin analogue) appears to be an attractive agent to combine with paclitaxel. The combination causes less nausea and vomiting, less neurotoxicity and can be administered to outpatients. Current studies define the role of carboplatin, doxorubicin, gemcitabine and new drugs such as topotecan in combination with paclitaxel. There is a renewed interest for the intraperitoneal use of drugs but this route of administration remains investigational.

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The management of recurrent epithelial ovarian cancer

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Recurrent epithelial ovarian carcinoma, i.e. relapse after a disease-free interval of at least 6 months following primary therapy, carries a poor prognosis, and the shorter the disease-free interval the worse the outcome. There has been considerable differences regarding the management, particularly, the role of surgery. In principle, recurrent disease has rarely, if ever, been considered as localized disease. Thus, it requires systemic therapy. Whether or not surgery has a place in combination with chemotherapy is still controversial. Clearly, if the recurrent tumor does not respond to chemotherapy surgery has little to offer, and is of palliative treatment. Should patients with recurrent ovarian carcinoma undergo cytoreductive surgery followed by chemotherapy as with primary disease? Alternatively, should they have induction chemotherapy, and only the responders be treated with interval surgery followed by further chemotherapy? These are some of the question that yet to be determined. Patients who initially responded to cisplatin based chemotherapy may well be treated again with such regimen. A response rate of 60% can be expected.

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Genetic predisposition to multiple cancers

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Genes predisposing to cancer in childhood may be responsible of multiple tumours. P53 germline mutations account for a substantial part of second malignant neoplasms (SMN) after a first cancer in childhood. Among 33 patients treated in the Department of Paediatrics affected by a SMN and tested for p53, 8 (including 2 sibs) were found to be carriers of a germline mutation of this gene. Most of these cases displayed a family history suggestive of LFS. One case was due to a *de novo* mutation.

All the genes predisposing to cancer in childhood and possibly to SMN have not yet been identified and the observation of familial aggregation may be a good indicator of such genes. Some of them might interact with radiotherapy and chemotherapy which have been implicated in SMN occurrence. We instigated a case-control study (25 cases of SMN after a childhood cancer and 96 controls with no SMN) to evaluate the possible effect of unknown genetic factors, evaluated from familial aggregation, on the risk of SMN, and their potential interaction with the effects of treatment. We found an independent effect of both radiotherapy and family history on the risk of SMN, even after exclusion of cases with p53 mutations and Recklinghausen's disease. These results strongly suggest that other genes than the ones identified to date have to be looked for and that the follow-up of children treated for a cancer should take account of genetic predisposition.

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The role of therapy in the incidence of second cancers

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Purpose: Recent UK data suggest at least 60% of children with cancer are cured. Therefore 1 in a 1000 of the general population will soon be survivors of childhood cancer. What are the risks of carcinomas among these adult survivors? Are there particular elements of therapy for childhood cancer which increase the risk of carcinoma development?

Methods: A cohort of 13279 patients who survived at least three years after diagnosis of childhood cancer between 1940 and 1983 was established using the population-based National Registry of Childhood Tumours. A case-control study was also established: cases were patients developing carcinoma and up to 4 controls were matched to each case. Cumulative doses of radiation and chemotherapy were compared between cases and controls.

Results: 69 carcinomas were observed in the cohort, 25 skin and 44 of other sites. By 30 years from three-year survival 2.5% of patients had developed a carcinoma, 1% of skin and 1.5% of another site. There were 12, 9, 9 and 8 cancers diagnosed in digestive, breast, thyroid and genitourinary tissue, respectively. The risk of carcinoma increased with increased exposure to radiation. Patients whose tissue had received 20–30 Gy and at least 30 Gy experienced 18 and 12 times the risk associated with unirradiated tissue, both of these relative risks being associated with $p < 0.001$.

Conclusions: These data have implications for monitoring patients treated in the past, and for planning future treatment protocols to achieve an optimum balance between the risks and benefits of different elements of treatment in the long-term.

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Growth from child to adult – Interference by radiotherapy

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The advances in treatment of childhood malignancies is reflected in improvement of survival. But the results are marred by delayed sequelae caused by the inability of radio- and antineoplastic therapy to discriminate between normal and target tissue. Irradiation to a growing child interferes with tissue growth. Radiation damage of the skin and the subcutaneous tissue and muscle can be serious. Radiotherapy to the thorax in a pre-pubertal female causes atrophy of the breast. Radiation to the small and large intestines