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Book Review

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Cancer Care in the Community & Cancer Care in the Hospital Edited by B. Hancock, Radcliffe Medical Press, Oxford/New York, 1996

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BOTH BOOKS are written by a dozen of contributors from Sheffield, U.K. and reflect a convincing 'unité de doctrine'. They are advertised 'to provide a comprehensive and concise review of all aspects of cancer management'. Do they achieve this high goal?

The first volume about 'Cancer Care in the Community' consists of 14 short chapters on basic science, epidemiology and education, screening and prevention, management and supportive care, all very well written by members of the Sheffield oncology team. Teal highlights are the chapters on palliative and supportive care, ethical issues, communication and psychological aspects—all representing extensive personal experience in these aspects of cancer care. I feel that this handy book is a rewarding read, especially for those not permanently working with cancer patients, but also for 'old pros'. The volume could be of great help to all involved in cancer care, from students and nurses to specialists and public health officials.

The second volume on 'Cancer Care in the Hospital' seems to be to be less convincing. It should advocate modern medical practice with special attention to up-to-date details. However, many chapters are just too short to really achieve this goal. Most are rather incomplete and superficially written, i.e. those on staging and investigation, on other treatments, on breast and colorectal cancer. Others, like those on gynaecological and genitourinary cancers, are of more help and provide valuable information about what to do in a specific hospital situation. I thought the book lacked a common chapter for the management of all tumour entities. Cancer Care in the Hospital may satisfy as an introduction to oncology for nursing students and lay persons, but might be less helpful for personnel in the field of oncology. Those looking for more extensive information should turn to other textbooks of oncology.

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Letters

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Comments on *Current and Future Trends in the Multidisciplinary Approach for High-risk Breast Cancer. The Experience of the Milan Cancer Institute*, Bonadonna, *Eur J Cancer*, 32A, No. 2, pp. 209–214, 1996

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I READ with interest the recent paper by Bonadonna [1], in which he comments on angiogenesis in breast carcinoma and states that "It is quite possible that human breast cancers also produce a specific angiogenesis inhibitor, which, like an endocrine hormone, inhibits growth of micrometastases."

The two animal studies Bonadonna mentioned were conducted by Gündüz, Fisher and Saffer [2, 3]. Their first study described the kinetic changes occurring in residual tumour following removal of a transplantable C3H mammary tumour [2]. The observed kinetic changes were associated with more rapid growth of metastases. The increased cellular proliferation occurring in metastases has received little attention, despite the potential importance of this observation. The increase in labelling index was due to non-cycling cells becoming proliferative and, therefore, more vulnerable to cytostatic agents. The second study was carried out to determine how a variation in the time interval between primary tumour removal and administration of a single dose of cyclophosphamide affected labelling indices of residual tumour cells and their growth [3]. The greatest effect occurred when cyclophosphamide was given prior to surgery. It completely prevented the increase in labelling index resulting from tumour removal and more effectively suppressed the growth of residual tumour. These results provided a biological rationale for the use of peri-operative adjuvant chemotherapy.

We recently analysed data from 370 patients with metastatic breast carcinoma [4]. Of interest, first metastases were

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predominantly found in visceral sites in patients having radical or modified radical mastectomies. The propensity of visceral metastases in patients having radical or modified mastectomies in our study is of concern as this is not a common finding. A former report by Valagussa and colleagues emphasised that the sites of first relapse after radical mastectomies were documented to occur preferentially in distant organs and tissues [5]. In another large series, locoregional recurrences and distant metastases were present in 24% and 49% of patients, respectively, 10 years after a radical mastectomy [6].

In the light of the aforementioned studies, a question arises: does surgery enhance the spread of metastases in breast carcinoma? Metastatic capacity depends in part on angiogenesis, in which tumours induce the formation of new blood vessels which provide nutrients for tumour growth and create access to circulation for metastasis. If breast tumours are secreting potent angiogenesis inhibitors and this secretion ceases after surgery, the surgery is a real challenge for metastatic spread. However, to penetrate the extracellular matrix, the metastatic cells need to disrupt the basement membrane with proteinases. One could speculate that surgery may augment disruption of the basement membrane. Once tumour cells enter the stroma, they can gain easy access to lymphatics and blood vessels for further dissemination. Currently, we do not know the correct answer, but future studies are awaited to clarify the effect of surgery on metastases in breast carcinoma.

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13-*Cis*-retinoic Acid and Alpha-interferon in Advanced Squamous Cell Cancer of the Oesophagus

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RETINOIDS, a class of compounds related to vitamin A, normally play a role in growth, vision, epithelial cell differentiation, and immune function [1]. A preventive effect of vitamin A on the development of chemically induced tumours has been demonstrated, as well as a therapeutic effect in cancer [2]. Cytokines, such as interferons, have demonstrated a synergistic effect with retinoids on the inhibition of proliferation in squamous cell carcinomas of the cervix, head and neck, and skin [3]. Here, we report the results of a phase II study of 13-*cis*-retinoic acid plus interferon alpha-2A in patients with metastatic squamous cell carcinoma of the oesophagus.

All patients were required to have measurable disease; age $\leqslant 75$ years; no prior chemotherapy; performance status (WHO) 0–2; life expectancy >3 months. Treatment consisted of interferon alpha-2A (IFN-α, Roferon-A^R, Roche) s.c. 3×10^6 IU every day, plus 13-*cis*-retinoic acid (cRA, isotretinoin, Roaccutan^R, Roche) orally 1 mg/kg/day. Treatment was continued for at least 2 months in all patients, unless disease progressed earlier, and for at least 3 months in case of no change, unless toxicity was intolerable. Response and toxicity were evaluated according to WHO criteria.

10 patients entered the study, all evaluable. The median duration of treatment was 8 weeks (range 4–33 weeks). One patient discontinued treatment after 4 weeks because of overt progressive disease. The patient characteristics are shown in Table 1.

No or very mild nausea (WHO grade 0, 1) was present in 9 patients, grade 2 in 1 patient. 2 patients developed grade 1 leucopenia, and nearly all patients showed a slight but discernible decrease in platelets, but still within the normal range (WHO 0). In 1 patient, the dose of cRA was reduced to 0.5 mg/kg/day because of difficulty in swallowing, and a dry and moderately painful skin. A dry skin was noted in 8 patients (WHO 1). Fatigue was seen only during the first 1 or 2 weeks after the start of treatment. No elevations of serum transaminases could be detected.

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