5th International Conference on Adjuvant Therapy of Primary Breast Cancer

This conference will be held in St Gallen, Switzerland between 1–4 March 1995. The deadline for abstracts is 31 October 1994. For more information, contact Mrs B Nair, c/o Professor H J Senn, Department of Medicine C, Kantonsspital, CH-9007 St Gallen, Switzerland. Tel. 41 (0) 71 62 10 97; Fax 41 (0) 71 25 68 05.

19th LH Gray Conference: Quantitative Imaging in Oncology

This meeting will be held between 3–4 April 1995 in Newcastle, U.K. Papers and posters are invited on diagnosis and quantitation of disease, techniques for treatment volume definition, uses of various imaging modalities in treatment planning, treatment planning systems—the state of the art and its current potential value, treatment verification and quantitative assessment of tumour response. It is anticipated that the meeting's proceedings will be published as a peer-reviewed supplement of a leading scientific journal. For further information, contact Dr K Faulkner, Regional Medical Physics Department, Newcastle General Hospital, Westgate Road, Newcastle-Upon-Tyne NE4 6BE, U.K. Tel. (091) 273 8811, ext. 22400; Fax (091) 226 0970.

Third Central European Lung Cancer Conference

This meeting on recent developments and controversies in lung cancer will be held in Prague, Czech Republic on 28–31 May 1995. For further information, contact the Conference Secretariat, Czech Medical Association, J E Purkyne PB 88, Sokolská 31, 120 26 Prague 2, Czech Republic. Tel: (42) 2 296889/297271; Fax: (42) 2 294610/24216836.

European Cancer Centre/NDDO Postgraduate Course on Basic and Current Topics in Anticancer Drug Development

This will be held on 7–8 September 1995 at Vrije Universiteit, Amsterdam, The Netherlands. For further information contact the European Cancer Centre, c/o AZVU, PO Box 7057, 1007 MB Amsterdam, The Netherlands. Tel. 31 20 644 4500/4550; Fax 31 20 644 4551.

The Sixth International Conference on Topoisomerases: From Molecular Aspects to Clinical Application

This conference, organised by the European Cancer Centre and NDDO, will be held on 1–4 October 1995 at Vrije Universiteit, Amsterdam, The Netherlands. For further information contact the European Cancer Centre, c/o AZVU, PO Box 7057, 1007 MB Amsterdam, The Netherlands. Tel. 31 20 644 4500/4550; Fax 31 20 644 4551.

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Letters

Vasectomy and Testicular Cancer

K. P. Dieckmann

IN AN ISSUE of this journal (Vol. 29A, No. 7), much space was devoted to the question of whether vasectomy can increase the risk of testicular germ cell cancer (GCT). While the epidemiological data presented [1] are hardly misleading, the take-home message for the reader remained somewhat less clear-cut.

Our own data clearly support the contention that there is no increased risk for GCT after vasectomy: in a case–control study, 4 patients among 538 patients (0.74%) treated for GCT during the period 1969–1990 in five Berlin-based urology departments were found to have had previous vasectomy. In the control group which consisted of 531 otherwise healthy age-matched males treated for various injuries in the same hospitals, 4 men also reported a previous vasectomy (0.75%). The relative risk (RR) thus amounts to 0.98 with 95% confidence intervals 0.24–3.96, indicating that there is no significant association between the two conditions ($P = 0.736$, χ^2 test).

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Whether or not these data and similar data of Morris Brown and colleagues [2] that were not mentioned in the article [1], are added to the meta-analysis of Lynge and colleagues [1], there was no need to formulate the conclusions as cautiously as the authors did. In fact, there is overwhelming epidemiological evidence that vasectomy does not have a bearing upon the pathogenesis of testicular GCT.

In addition, the biological explanation of why vasectomy could at least accelerate the occurrence of GCT as offered by Jorgensen and colleagues [3] is a rather mechanical view on the pathogenesis of GCT. While it is undisputed that all GCT are derived from testicular intraepithelial neoplasia (TIN; carcinoma *in situ*), the factors influencing the transition of TIN to frank GCT are unknown [4]. However, it is much more probable that molecular genetic or immunological [5], or endocrinological factors [6] are involved rather than simple increase of fluid pressure within the rete testis and seminiferous tubules. Even if this hypothesis was true, it would not account for an overall increase of GCT in the group of vasectomised men.

In summary, epidemiological studies have failed to document an association of GCT and vasectomy [7]. The only study that indicated an increased risk showed a relative risk of 4.2 which is not a convincingly high factor, as demonstrated by wide 95% confidence intervals of 1.8–8.2. Thus, a selection bias might