Letters to the Editor

An evaluation of the Technicon AutoAnalyzer for automating complement-fixture tests

The authors (Taylor et al, J. clin. Path. 1968, 21, 521-526) have tried to automate the complement-fixture test without taking advantage of the many previous studies published in England since 1964 (Irving, 1966; Pugh, 1967), in Canada (Reed, Prytula, and Smith, 1967), and in France (Gaillon, Ripault, Studi6vic, and Dausset, 1966; Studi6vic, 1965; Vargues, Studi6vic, and Ripault, 1965a; Vargues, Studi6vic, Moraud, and Gonthier, 1965b).

In fact, automation has been realized since 1964 and has been the object of many papers by Vargues et al (1965a and b), Gaillon et al (1966), Irvine (1966), Reed et al (1967), Valette and Joubert (1967), Roumiantzeff (1967), and ourselves (Vargues et al, 1965a and b; Studi6vic, 1965).

The automated complement-fixture test (WR) is used in systematic routine applications in a large number of laboratories with cardiolipidic or treponemic antigens. Carryover from positive samples to negative specimens is non-existent and there are no wash problems with the continuous flow-cell. Several hundreds of specimens can be easily handled every day without special difficulties.

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References

The problem of ‘chronic mastitis’ with epitheliosis

The May number of Clinical Pathology (pp. 340-347, 1969) contains an article by J. B. Macgillivray with the above heading.

Dr Macgillivray finds that the incidence of invasive cancer after local excision of breast tissue showing chronic mastitis with epitheliosis was nil. He demonstrates the obvious discrepancy compared with my series, in which nine out of 20 with this special form of chronic mastitis developed cancer.

I agree with Dr Macgillivray that allowances have to be made on account of his smaller series and shorter average follow up (seven years versus 17 years), but I doubt whether he is right when he suggests that another possible factor in the high incidence of malignancy in my series should be in the inclusion of cases which, in reality were lobular carcinoma in situ or intraduct carcinoma from the start. He cannot exclude that a few of my cancer cases contained small foci of lobular carcinoma in situ combined with several epithelioses but the manifest cancers were not of the invasive lobular carcinoma type. As for the possibility of my including cases of intraduct carcinoma, this is highly improbable. I have not only excluded borderline cases (and I agree with Dr Macgillivray’s definition) but we have also excluded cases which other pathologists labelled as epithelioses, but which in my eyes might be suspected as borderline cases. Other arguments for not having included intraduct carcinoma are mentioned in my monograph.

Another explanation of the discrepancy might be that Dr Macgillivray’s follow up only comprises 50% of the patients. If I had been satisfied with this percentage, I should nearly have come to the same conclusions as Dr Macgillivray. However, I found most of the cancer cases in the next 45%, a group which was more difficult to trace.

I look forward to Dr Macgillivray’s continued studies with bigger series, longer observation periods and—above all—with a better follow up. Until then, I find no reason to doubt that severe epitheliosis involves a considerable risk of developing cancer of the breast.

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References

Calculation shows that nine tumours in 340 total years of follow up in Kier’s series is on the incidence of one per 38 years which should be compared with no tumours in 49 years of follow up (Macgillivray, 1969). No statistical test is needed to appreciate the lack of a significant difference.—ED.