TABLE 1 *Observed and expected cases of lung cancer occurring in barbiturate users, as related to receipt of benzodiazepines or of more than one prescription of barbiturates*

	Number receiving and at risk	Cases		Standardized morbidity ratio (SMR) [95% confidence limits]	Sex-specific SMR
		Observed	Expected		Males
Barbiturates overall	10 127**	107	64.5	1.7 [1.4, 2.0]	1.6
Barbiturates—no benzodiazepines	6611**	53	40.0	1.3 [1.0, 1.7]	1.4
Barbiturates and benzodiazepines					
Any interval*	3505**	43	21.8	2.0 [1.4, 2.7]	1.7
Within 30 days	708	11	4.5	2.4 [1.2, 4.4]	1.6
Same day	326	6	2.1	2.9 [1.0, 6.2]	2.2
Barbiturates at least twice	1414	33	11.3	2.9 [2.0, 4.1]	2.5
Barbiturates at least three times	317	12	2.5	4.8 [2.5, 8.4]	3.4

*Up to four years; drug data collected July 1969–August 1973.

**Discrepancy between total barbiturate users at risk (10 127) and sum of those also receiving (3505) and not receiving (6611) benzodiazepines is because 11 users of both drugs developed lung cancer after receiving a barbiturate but before receiving a benzodiazepine.

the lower 95% confidence limit now reaching 1.0. In view of the emphasis by Anthony *et al* on the simultaneous use of barbiturates and benzodiazepines we attempted to assess simultaneity by restricting the interval between dispensing of the two types of drugs. (We do not know, of course, exactly when the drugs were actually taken, only when they were received.) When we required that the interval be within 30 days the SMR rose to 2.4 and when we required that both types of drugs be dispensed on the same day the SMR rose further to 2.9. These restrictions led, of course, to drastic drops in the numbers of subjects at risk available for study. In contrast to the findings of Anthony *et al* women taking both types of drugs showed a greater relative risk of lung cancer than did men.

To place these findings in better perspective we repeated the analyses, starting with the entire barbiturate group but limiting follow-up to those who received barbiturates at least twice or at least thrice, regardless of interval. With the former restriction of SMR increased to 2.9, and with the latter to 4.8. These findings, suggesting a dose-response relation, appear to contradict our previous chart-review data in this respect.

Although Anthony's findings on the combined effects of barbiturates and benzodiazepines are not refuted by these data, they are weakened by the ability of two dispensings of barbiturates to predict lung cancer more accurately than the combination of barbiturates and benzodiazepines. My own best guess is that taking both barbiturates and benzodiazepines or receiving multiple prescriptions for barbiturates is a marker of some other behavioural characteristic closely associated with lung cancer, most likely smoking larger numbers of cigarettes, avid inhalation of smoke or some other characteristic implying more intense exposure to tobacco smoke. Also, alcohol consumption is often high in users of these drugs and may be related to lung cancer, possibly interacting with cigarette smoking.[3] I believe that this type of explanation must be ruled out before either barbiturates alone or the combination of barbiturates and benzodiazepines can be implicated as causal factors for lung cancer.

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From J H Abramson*

Broadening the Scope of Clinical Epidemiology

Sir—The scope of clinical epidemiology—or, for those reluctant to recognize clinical epidemiology as a distinctive domain, the scope of the use of epidemiology in clinical settings—is primarily determined by the needs of clinicians. Its central functions are to improve clinical care and the use of the clinical situation for research

These are the aspects on which textbooks of clinical epidemiology[1,2] concentrate.

A new development in clinical practice, which calls for a considerable broadening of the scope of clinical epidemiology, is the growing interest in community-oriented primary care (COPC), a term introduced by Kark.[3] In the new dictionary of epidemiology COPC is defined as 'an integration of community medicine with the primary health care of individuals in the community. In this form of practice the community practitioner or community health team has responsibility for health care both at a community and at an individual level'.[4] This kind of practice is based on epidemiological appraisal of the community's health needs and the establishment of programmes, in the framework of primary care, to meet these needs.[3,5,6]

There is increasing awareness of the role that COPC can play in improving the health of populations. Its potential value is probably most obvious in developing countries,[7] but in developed countries also, its effectiveness in dealing with hypertension and other major health problems has been demonstrated.[8–10] Recent symposia describe the use of COPC in various settings in different countries.[11,12] In the United States, a conference was recently held under the auspices of the Institute of Medicine of the National Academy of Sciences to review current thinking on COPC and make proposals for its development in that country.[13–15]

In COPC, epidemiology not only gives clinicians a rational basis for decisions on the diagnosis, prognosis and treatment of patients (as in other forms of clinical practice), it is also an indispensable tool for the 'care of the community as a patient'.

If the scope of clinical epidemiology is to be widened to embrace the requirements of COPC, it is not enough merely to add the methodology of community surveys to the topics already covered. Consideration must be given to the uses of epidemiology in each phase of the COPC process: in the preliminary phase, during which the community is defined and described and its main health problems are identified; in the phase of community diagnosis, when selected problems are investigated in some detail; in the planning of intervention; in the implementation and monitoring of programmes; in ongoing surveillance; and in evaluation. These uses of epidemiology are described elsewhere,[5,16] with illustrative material from the practice of the Hadassah community health centre in Jerusalem, which has demonstrated and taught COPC since the 1960s.[3]

Moreover, a number of specific issues that arise when epidemiology is applied in the COPC context need careful consideration. These include problems in the definition of the target community, methods of demographic surveillance, the design of primary care records and record systems, the identification of community health syndromes, the use of quasi-experimental methods in programme evaluation, and many others.[17]

COPC presents epidemiologists with at least three roles:

1. As trainers of present and future practioners.[5] All COPC practitioners require enough exposure to epidemiology to enable them to appreciate its importance, play their part in the collection of accurate data, and make appropriate use of the findings.
2. As participants in the practice. Epidemiologists should be prepared to become involved in supportive or consultative roles, or as partners, especially in demonstration and teaching practices. One of the proposals for the furtherance of COPC in the US is the formation of a network of primary care practices that are affiliated with academic centres, to develop COPC principles and produce COPC practitioners.[18]
3. As researchers, with emphasis on developing and testing tools and techniques for use in community diagnosis, community health surveillance and programme evaluation, and on evaluative studies.

Epidemiologists who believe that COPC is a good thing can promote it by performing the above functions. In an analysis of obstacles to the spread of COPC, Rogers concludes that among others there is an urgent need 'to develop a more vibrant and compelling data base with which to make a case for COPC, and train a small cadre of people to do it well'.[15]

It may be asked whether clinical epidemiology can retain its distinctiveness if its scope is widened to include the additional elements suggested above. Opinions will differ. But whether we speak of clinical epidemiology or only of the application of epidemiology in clinical settings, the needs of community-oriented clinical practice must be met.

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From Bengt Källén*

Modelling Maternal Birth Cohort Effects on Malformation Rates—a Reply

Sir—In a Letter to the Editor,[1] Harris and Mathers commented upon mine and Lindham's[2] study on gastroschisis and maternal birth cohort effects. The comments seem to concern a paper other than the one we wrote. We did not, in fact, try to determine the effect of maternal birth year and age on the gastroschisis rate in the way Harris and Mathers believe and criticise. We described changes actually observed in two parameters: prevalence at birth of this malformation and mean maternal age. 'In order to study the possibility that the observed changes *could* be explained by a maternal birth cohort effect, such effects were simulated. . . .' (p. 399). We then found that the changes observed could indeed be simulated in this way and that the expected numbers obtained were reasonably similar to those observed, and that changes in maternal age distribution were also simulated in a way which resembled the changes actually observed. This does *not* prove that our model is true—and we have no intention of estimating coefficients which can describe a relationship between maternal birth year, maternal age, and the risk of having a baby with gastroschisis the way Harris and Mathers apparently think, and absolutely no thought of applying a chi-square test for heterogeneity between the 272 'cells' of the material! The numbers are much too small to permit any extravagant statistical calculations.

If the hypothesis presented by us (and we did stress that it was a hypothesis) is true, it might be applicable to other data on women who had infants with gastroschisis in a population where an increase in prevalence at birth of this malformation has been noted. Perhaps the Australian National Perinatal Statistics Unit (where obviously more births must be registered than in Sweden) has adequate knowledge of the presence of malformations in infants to test the hypothesis on their own data? That the increase in prevalence of gastroschisis is not a unique phenomenon for Sweden is apparent from the recent 1981 Annual Report of the International Clearinghouse for Birth Defects Monitoring Systems.[3] Another way to test the hypothesis is to use it to predict what will happen in Sweden during the 1980s. So far, too few years have elapsed—but data for 1981–82 actually fit well with the model presented.

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