should be aware of the whole spectrum of possible causes of colonic inflammation—infective as well as non-infective ones.

We are most interested in their series of patients with "infective diarrhoea" who had negative stools. For some years now, in collaboration with Dr B C Morson and his colleagues at St Mark's Hospital, London, we have been studying patients presenting with bloody or persistent diarrhoea and we have become aware of a number of patients who cannot be categorised into any of the recognised types of colitis. For want of a better term we are using the name "transient colitis" to describe such cases. These patients present with diarrhoea (frequently bloody) of sudden onset, with the sigmoidoscopic and rectal biopsy changes of acute colitis and consistently negative stool cultures. The outcome is invariably satisfactory: the symptoms subside within a few days and follow-up so far has revealed no instance of a relapse. In 172 patients with diarrhoeal states there were 34 patients who fell into this category. Histologically, some of these patients had the appearances of infective colitis, but others showed changes suggestive of IBD. We tend to take the view that these patients were suffering from an infective colitis, the negative bacteriology being due either to prior antibiotic therapy or to infection with organisms other than shigellae, salmonellae, or amoebae. The recent discovery of some campylobacter species as significant enteric pathogens is of considerable interest and may have accounted for some of these cases. A quarter of our patients with campylobacter diarrhoea had bloody diarrhoea and we have found evidences of colitis at sigmoidoscopy and biopsy in a number of these patients. It is interesting that four of these patients had rectal mucosal changes highly suggestive of IBD.

It seems likely that many patients labelled as having ulcerative colitis or colonic Crohn's disease on sigmoidoscopy and biopsy findings with negative bacteriology, but who have remained in complete remission since the initial attack, are in fact examples of "transient colitis," probably of infective aetiology. Therefore we fully endorse the view of Dr Dickinson and his colleagues that the diagnosis of IBD should not be made prematurely in view of its long-term therapeutic and social implications.

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Time and the consultation in general practice

SIR,—Having read the interesting report by Dr K B Thomas (7 October, p 1000) I think his data capable of an alternative explanation. The majority of patients of the kind detailed in the study seen in general practice do not return within one month—they have self-limiting symptoms which disappear with or without, because of, or in spite of treatment. This is clearly shown in the table. Some patients do return, however, and it seems on examination of the data that there is a reduction

of over 50% in patients who returned with the same or a different complaint in the patients in the "long no treatment" group. I postulate that Dr Thomas's results show that there are two groups of patients—the majority who attend once and a minority who do benefit from care and attention and a reasonable explanation of their symptoms.

How can we recognise this minority? Until we do so I think that it will be very difficult to prove anything about the length of time of consultations, as data will be diluted by those for whom time does not seem to be a factor.

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SIR,—Dr K B Thomas in his report "Time and the consultation in general practice" (7 October, p 1000) is using a statistical device designed to make sure that a positive association is not shown to exist when none does exist. In other words it errs on the side of caution. The study does not prove that there may not be a positive association between length of consultation and number of return visits a month later—merely that none has been proved with the number of patients studied.

Taking the last two columns and lumping "the same complaint" and "different complaint" together we find that with "short no treatment" 13 out of 50 return and with "long no treatment" 6 out of 50 return. Statistically there is no significant difference. However, if the trend of difference were continued to 100 patients the following figures emerge: with "short no treatment" 26 out of 100 return and with "long no treatment" 12 out of 100 return. The difference now becomes statistically significant, favouring "long no treatment."

There are other criticisms of his paper. Firstly, taking the last two columns again, it may be expected that "short no treatment" would not encourage patients to return—the patient might say to himself, "Why bother? He was completely unhelpful." Thus "short no treatment" might contain a group who did not return because of dissatisfaction. Secondly, Dr Thomas uses return visits to measure outcome. Surely the patient's view of the consultation must be part of the measure of outcome. Rating scales are available for this purpose.

It would have been easier to accept Dr Thomas's report if he had been more cautious in his conclusions. The subject still awaits clarification.

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Treatment of migraine

SIR,—Dr David Thrush (7 October, p 1004) states that "referral to the psychiatrist rarely helps [in the treatment of migraine] and may unmask yet more problems." Nevertheless, he considers that "successful treatment depends on correct diagnosis, treating the patient as well as the disease, and spending time talking to the patient—a most neglected therapeutic weapon."

Had he included in his list of essential prerequisites to successful therapy the need to provide a ready ear to the patient's statements he would have clearly indicated his respect for the psychiatric treatment "which rarely helps." He suggests that patients who are

refractory to treatment "are best treated with psychotherapy from the general practitioner, who knows both the patient and his background well"—surely an acknowledgment of the value of psychiatric treatment even if practised by the GP.

Certainly referral to a psychiatrist "may unmask yet more problems," but surely this is very necessary in the management of a painful and disabling entity of obscure aetiology notoriously unresponsive to drugs and frequently precipitated by neurotic factors. Dr Thrush is more psychiatrically minded than he appreciates and should not be reluctant to refer his migraine patients for psychiatric investigation. The outcome may surprise him.

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SIR,—In his article on the treatment of migraine in *Personal Therapeutics* (7 October, p 1004) Dr David Thrush says that ergotamine has never to his knowledge been shown to be more effective than a placebo in a double-blind trial. Although oral ergotamine has not been shown to be better than a placebo in some trials,^{1,2} it has in a sequential trial which used a 5 mg oral dose.³ Inhaled ergotamine, which Dr Thrush does not find helpful, has been shown to be superior both to an inhaled placebo and to a sublingual preparation.¹

There is considerable difficulty in evaluating the place of ergotamine in the treatment of migraine. Preparation, route, timing, dose, and selection of patients may all be important. It seems to me unlikely that clinical experience will sort out all these variables and further randomised trials are desirable. The discrepancies in the literature, and the paucity of evidence from randomised trials, on the value of ergotamine are surprising in a condition as prevalent as migraine.

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¹ Crooks, J, Stephen, S A, and Brass, W, *British Medical Journal*, 1964, 1, 221.

² Waters, W E, *British Medical Journal*, 1970, 2, 325.

³ Ostfeld, A M, *American Journal of Medical Science*, 1961, 241, 192.

Drug treatment of psychiatric patients in general practice

SIR,—May I please be allowed to make the following points following Dr Peter Tyrer's article (7 October, p 1008)?

Academic medicine, pharmacological principles, etc, etc, are a means to an end, not an end in themselves. What matters is that patients get better, and in these times of scarce resources the greatest good is done with the least cost. General practitioners all have a horde of chronically depressed and anxious patients who are a constant source of work and difficulty. These patients require regular support and often regular drug treatment, without which their lives (and ours) are made a misery. Dr Tyrer suggests (he by no means proves) that they are being incorrectly prescribed to by their general practitioners. In whose opinion, pray? Dr Tyrer has judged that the various treatments are incorrect "on pharmacological grounds," but did he ask the patients how often their GPs had tried to get them off medication only to be pleaded with to let them continue with their treatment?

The proof of the pudding is the fact that

there are innumerable patients who are managed quite well by their GPs and who never go near a psychiatrist, nor do they want to. You stick to your neat and cosy theoretical principles, Dr Tyrer. We are doing the work under difficult circumstances and with very little time for each patient. I have yet to refer a patient to a psychiatrist without his being prescribed a vast amount of drugs before the patient got better or more manageable.

Let's have less "GP baiting" from the ivory towers of academic excellence and more practical help and advice on how to help our patients. Anyone can criticise anything or anyone. It takes someone of real worth to suggest something better or get the job done any better.

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Perforated duodenal ulcer on diflunisal (Dolobid)

SIR,—We read with interest the letter from Drs H P Upadhyay and S K Gupta (26 August, p 640) reporting an overdose of diflunisal. Recently we treated a patient with a perforated, acute duodenal ulcer while on treatment with diflunisal (Dolobid). Prior to starting treatment he had no gastrointestinal symptoms. He was prescribed two tablets twice daily for pain in his knee. He took no other medication. On day 2 of treatment he started to feel epigastric pain and his ulcer perforated on day 7.

The manufacturers' information booklet records one associated duodenal ulcer. They report a series in which faecal blood loss was significantly lower on diflunisal than on a comparative dose of aspirin.¹ However, the blood loss on diflunisal was significantly greater than on a placebo. It is our clinical impression that a large number of patients presenting to hospital are already being treated with diflunisal. Will it continue to be as trouble free as predicted?

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¹ De Schepper, P J, *et al*, *Clinical Therapeutics*, 1978, 5, suppl A, p 49.

Death certification and epidemiological research

SIR,—Sir Cyril Clarke concludes the report entitled "Death certification and epidemiological research" (14 October, p 1063) by saying "The purpose of this paper is to try to improve the accuracy of the information which the OPCS receives," but the study group and the report have overlooked so many pertinent points that they have vitiated their good intentions.

In the first place there is no reference to the Brodrick report, *Death Certification and Coroners* (Cmnd 4810, 1971), which deals far more effectively with the problems which they encountered and makes fuller recommendations.

Secondly, if coroners are to be criticised, even constructively, would it not have been a good idea first to consult a coroner, or better still the Coroners' Society, to find out why coroners do not consult the clinician in charge more often? Would it not have been a good

idea also in cases 4, 5, 6, 12, 13 and 14, which were clearly the subjects of inquests, to find out something of the circumstantial evidence considered by the coroner before reaching his verdict as incorporated in the death certificate? If this was done there is no indication in the paper that it was, but rather we are given the picture of a group of omniscients, looking only at the medical notes and deciding, without reference to the legal requirements of a verdict of "suicide," that they knew best. In case 6, for example, the words "He killed himself" are the formula to denote suicide laid down by the Coroners Rules 1953—and in fact the death certificate says precisely the same thing as the so-called "Actual cause of death," but it says it rather more fully.

Sir Cyril has treated very superficially a very important subject which has already been dealt with far more expertly by the Brodrick Committee. Reform is much overdue and it is a sad reflection on the Government's priorities that it can find the money to set up OPCS but not the parliamentary time to give it a proper death certification system on which to base its epidemiological research. In the meantime it would help if consultants took a greater interest in death certification, both by ensuring that their junior staff are adequately supervised and by giving as much information as possible to pathologists before they embark on necropsies, whether at the request of the coroner or not.

If the Royal College of Physicians wants better mortality statistics it must put its own house in order before telling the coroners what to do—and it could achieve most by directing its strictures at our political masters, in whose hands the matter has rested for seven years.

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SIR,—I should like to comment on the paper "Death certification and epidemiological research" by the Medical Services Study Group of the Royal College of Physicians (14 October, p 1063). Since the authors point out that the study was suggested by me, may I say that I do not entirely agree with their interpretation of the findings, in particular with what they call major errors.

Many doctors will still recall the surprise which greeted the news that a radiological picture when read twice by the same or other radiologist was interpreted inconsistently.¹ It was soon realised that diagnosis includes a subjective element which by its nature varies from time to time. Ways to minimise "error" were devised—for example, independent diagnosis by different clinicians or repeated "blind" by the same clinician. Indeed, the essence of statistical analysis lies in taking account of variation and error.

Death certificates are widely used for research and have not escaped scrutiny. The method and findings of this report are in some ways similar to those of previous studies, in which, it should be recalled, the main point was the difference between diagnosis in hospitals before and after necropsy.^{2,3} But are the implications for research and statistics not in danger of being exaggerated? Do the authors overlook the steps taken by experienced analysts to correct and to allow for errors? Surely yes. A close look at the tables purporting to show major discrepancies reveals that

some discrepancies are minor, that in others the correct "underlying" cause would have been coded, and that in yet others the discrepancies are not serious in the context of the uses of these records.

What are the alleged major discrepancies in these tables, how do they come about, and what is their effect on statistics? Firstly, doctors may conceal what they consider embarrassing conditions such as alcoholism. Secondly, coroners usually omit psychological antecedents when certifying suicide; and the legal distinctions between suicide, open verdict, and accidentally self-inflicted death do not necessarily accord with clinical opinion. Thirdly, sometimes coroners' pathologists do not use the standard form for certifying cause of death. Fourthly, the recording of pathological conditions may be in the wrong order, may contain redundant information, or may be wrong (as judged by subsequent scrutiny). These last errors may indeed be the result of relegating the job of certifying to the most junior doctor without guidance.

This is quite a list of flaws; but they affect a relatively small number of certificates, and the errors are not as serious for research and statistics as would appear at first sight. For example, the effect of wrong order is minimised by rules of coding and by analysing multiple causes; indeed, some of the examples of major discrepancies would have been correctly coded. The avoidance of ostensible stigma is systematic and recognised and, as a result, the effect can be measured in special studies, such as the study by Barraclough *et al* of the precursors of suicide⁴ and the OPCS study of deaths of alcoholics.⁵ Death certificates carry only summaries of complex disorders. They should not be expected to be always comprehensive; as long as they have the correct clues—for example, suicide or cirrhosis of liver—the details may be established at another level of investigation. Detractors of death certificate statistics in asking for all or none betray their main mistake: they view the information on certificates as though it is treated in isolation. It is not. In fact it is seen as only part of a network. The information is invariably analysed in conjunction with other records—as indeed in the study under discussion.

Death certificates are used mainly for analysing trends, for correlations (for example, with environmental hazards, times or places), and for surveys designed to test causal hypotheses retrospectively and prospectively. For trends and correlations most diseases are diagnosed consistently enough. Many studies in depth attest to their reliability for these purposes, a recent example being the studies on the rise and fall of asthma mortality.⁶ In retrospective surveys hypotheses are tested by selecting certificates with particular diagnoses, and finding out whether the deceased persons had been exposed to the suspected hazard. For these studies diagnoses are invariably checked by recourse to clinical notes and opinions, and the use of controls allows for the play of chance. A prospective survey (a growing practice) begins with records of persons exposed to an alleged hazard, and subsequently their deaths are noted together with the diagnoses. Again, mortality patterns are compared with those of standard populations subject to similar limitations of diagnosis.

That the use of death certificates for research is increasing is not surprising. They are mandatory for every death, quickly processed, and indexed to be available for analyses and surveys. What were referred to as major