discrepancies turn out to be mainly aberrations which are correctable in their wider context. Over the years death certification has improved with advances in medicine. More information is given and it is more precise. We should tackle administrative weaknesses such as failure of pathologists to state explicitly causes of death, failure to update after necropsy, and relegation of completion to junior doctors without instruction. Over the past 140 years these records (the results of everyday experience of doctors) have contributed a great deal to our knowledge of the health of the nation and to medical research. Of course, there is a continuous need to study their flaws—especially as part of practical research—as was done in the papers by the study group of the Royal College of Physicians. I trust that these papers will be read constructively, otherwise they may have a negative effect, the best becoming the enemy of the good.

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¹ Birkelo, C C, *et al*, *Journal of the American Medical Association*, 1947, **133**, 359.

² Heasman, M A, and Lipworth, L, in *Studies on Medical and Population Subjects No 20*, p 133. London, HMSO, 1966.

³ Waldron, H A, and Vickerstaff, L, *Intimations of Quality*. London, Nuffield Provincial Hospitals Trust, 1977.

⁴ Barraclough, B, *et al*, *British Journal of Psychiatry*, 1974, **125**, 355.

⁵ Adelstein, A M, and White, G, in *Population Trends* 6, p 7. London, HMSO, 1976.

⁶ Inman, W H W, and Adelstein, A M, *Lancet*, 1969, **2**, 279.

SIR,—The opportunity for clinicians to communicate their clinical interpretation of the cause of death (14 October, p 1063) to the coroner via the pathologist is already freely available at the time of necropsy. How many take the opportunity, or even fill in a meaningful request form? Direct communication between the coroner and the clinician is unnecessary if the clinician attends the necropsy, and this saves his and the coroner's time.

The relatively good correlation between death certification and hospital notes in cases where no necropsy has been performed proves only that clinicians can interpret their notes, but little about the true cause of death. What might a necropsy have shown? The poor correlation where hospital necropsies have been done can only mean that clinicians do not understand the reports they receive, or have not attended the necropsy.

The interpretation of this study must be that clinicians should attend necropsies and talk to pathologists, and that the falling necropsy rate should be reversed.

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P J H FLETCHER
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SIR,—The paper on death certification and epidemiological research (14 October, p 1063) appears to be misleading in several of its premises and conclusions, and it is disappointing to see it published in your journal at the same time as the excellent series of articles on epidemiology by Professor Geoffrey Rose and Dr D J P Barker.

In the discussion section it is rightly pointed out that the underlying cause of death

is taken as the last diagnosis under section I of the certificate; yet in tables II and III, purporting to demonstrate "major discrepancies" between recorded and "actual" cause of death, several examples appear where the underlying cause, so defined, is manifestly the same.

A more fundamental fault, however, lies in the implicit assumption that "actual" cause of death can be defined. The authors assume that the cause of death assigned by their consultant colleagues is always the true one, and those of the certifying doctors, coroners, and pathologists potentially erroneous. In the introduction reference is made to earlier papers, all of which have a different premise—namely, that the necropsy-supplied cause is the true one. What is being studied is, of course, another example of observer variation or bias, and it is fortuitous for the authors that this is explained clearly in the series by Rose and Barker to which they refer. The perplexed epidemiologist whom their paper presumably aims to assist will, of course, be well aware of this phenomenon. Meanwhile one can only recommend to the senior clinicians concerned that if they remain unconvinced that they too might err they should fill in the death certificate themselves.

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Acute stroke

SIR,—Hopefully the article by Dr Graham Mulley and his colleagues (7 October, p 994) will stop the use of dexamethasone in acute stroke. Hitherto, too many patients have been given too much dexamethasone for too long a period with the production of frightening side effects rather than benefit. It is good to see a useless treatment being debunked and perhaps this is an appropriate time to debunk a useless investigation—namely, the lumbar puncture. While in the trial situation, such as described by Dr Mulley, there may be a place for examining the cerebrospinal fluid, there is surely no justification for this procedure as a routine investigation. As 134 patients in one general hospital were admitted to this trial over a 13-month period, what a saving there would be on a national level if dexamethasone were not prescribed and lumbar punctures not performed.

A C YOUNG

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SIR,—The study reported by Dr Graham Mulley and others (7 October, p 994) seems to give a fairly definite answer, in the negative, to the question of whether or not the disability resulting from thrombotic stroke might be diminished by giving steroids. I should like to draw your attention to a similar study carried out under the auspices of the New Zealand Medical Research Council's Standing Committee on Therapeutic Trials. This study¹ used betamethasone instead of dexamethasone but in similar dosage and for the same period, although the number of cases suitable for analysis was smaller.

The results were even more discouraging than those of Dr Mulley and his colleagues, there being a significantly higher death rate among the betamethasone group than those treated with placebo injections. There would certainly not seem to be any indication for this

type of treatment in the generality of thrombotic strokes.

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¹ McQueen, E G, *New Zealand Medical Journal*, 1978, **87**, 103.

Maprotiline hydrochloride and grand-mal seizures

SIR,—We thought it would be helpful to doctors if we commented on the letter from Drs M J Hall and R I Russell (30 September, p 961) describing a patient who had a grand-mal seizure when taking maprotiline (Ludiomil).

Of course it is well known that tricyclic antidepressants as well as tetracyclic compounds can cause epileptic seizures in some patients.¹⁻³ While it is true that we have little knowledge of the exact incidence of seizures in patients taking maprotiline, this is also true of other antidepressant drugs which have been on the market for much longer. The whole subject was well reviewed recently by Trimble,⁴ who regards maprotiline, along with flupenthixol, viloxazine and nomifensine, as least likely to excite epilepsy in known epileptic patients. It is, of course, difficult to obtain entirely reliable data on the exact incidence of a side effect when the number of cases seen by one investigator is very small.

The dosage recommendation for Ludiomil is normally 25-150 mg daily (*Data Sheet Compendium*, 1978), and we would like to emphasise that higher doses are required only in exceptional cases. This is important because epileptic seizures are rare when the dose is below 150 mg. The patient described had her dose of maprotiline quadrupled to 300 mg and was also taking Ovranette, which could increase susceptibility to epilepsy if there was fluid retention (*Data Sheet Compendium*, 1978). Also, several years ago Mills⁵ advised the prescription of an anticonvulsant to any patient taking over 200 mg of a tricyclic antidepressant.

Be assured that we will continue to define, by studies currently under way, the epileptic potential of Ludiomil in the hope of advising doctors even better how to use the drug safely.

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¹ Betts, T A, *et al*, *Lancet*, 1968, **1**, 390.

² Dallos, V, and Heathfield, K, *British Medical Journal*, 1969, **4**, 80.

³ Escande, M, *et al*, *Encephale*, 1976, **11**, 133.

⁴ Trimble, M, *Epilepsia*, 1978, **19**, 241.

⁵ Mills, I H, *Practitioner*, 1976, **217**, 529.

Abnormal cerebrovascular regulation in hypertensive patients

SIR,—Dr D N W Griffith and others (9 September, p 740) report on a defective carbon dioxide (CO₂) response in the cerebral circulation of 10 middle-aged hypertensive patients. In a similar study published in 1976¹ we found essentially no difference in cerebrovascular CO₂ response between hypertensive and normotensive patients. The authors