cardiac infarction must be explained in part by the relative frequency of cardiac failure as the cause of death, for which no more can be offered in the CCU than in the general ward. It is, however, surely important not to discard the opportunity of effective resuscitation—the stakes are high even though the number of patients benefiting may be small.

It must also be remembered that the study was undertaken in two hospitals possessing CCUs and, as the authors concede, the CCU provides a valuable training ground for junior medical staff and nurses, so that it is reasonable to assume that the resuscitation in the wards in such a hospital would be better than where no CCU existed. Withdrawal of the CCU or failure to develop such a unit might well result in lower standards of resuscitation and create an entirely new clinical setting which would require further evaluation.

We believe that while patients with acute coronary episodes continue to be admitted to hospital it is mandatory to provide the highest standard of care possible. At a time when the availability of doctors and nurses on the ward seems ever-diminishing some form of CCU organisation would appear to be the only answer. We must at least ask that any future studies of this question should define carefully the nursing and medical staffing arrangements involved so that valid comparisons may be made.

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¹ Joint Working Party, *Journal of the Royal College of Physicians of London*, 1975, 10, 5.

Fatal agranulocytosis attributed to co-trimoxazole therapy

SIR,—The recent article by Dr W H W Inman from the Committee on Safety of Medicines (11 June, p 1500) concerning drugs which may be related to fatal bone marrow depression is of major interest and importance. In essence Dr Inman has carried out an uncontrolled study of drug use prior to the diagnosis of aplastic anaemia or agranulocytosis, using for his data source the records of the Office of Population Censuses and Surveys (OPCS). While one might argue about the validity of regarding any exposure to bone-marrow depressing agents in the year prior to diagnosis as being causally related to the diseases of interest, this may well be justified in the case of drugs such as phenylbutazone which are well known to cause bone-marrow depression.

However, Dr Inman's assertion that over half the fatal cases of agranulocytosis were caused by co-trimoxazole requires more careful scrutiny. Agranulocytosis is a condition which almost invariably is brought to light as a result of infection. Co-trimoxazole is one of the most frequently prescribed antibacterial drugs in the United Kingdom. Therefore one would reasonably expect a strong association between co-trimoxazole and agranulocytosis even in the absence of a causal relationship between drug and event. To prove a causal relationship either a proper case-control study would be required or alternatively much more information on the true relationships and use of other antibacterial agents would be required than has been given by Dr Inman. Thus we feel the case against co-trimoxazole as a common cause of agranulocytosis is as yet

unproved. Nevertheless, since there is some prior suspicion that the drug could cause this disease by virtue of the chemical structure of one of its components, further studies are obviously required.

We therefore sought from the OPCS details of the number of patients dying between 1960 and 1974 from all anaemias, aplastic anaemia, and agranulocytosis.¹ The numbers, divided into five-year periods, are shown in the accompanying table.

Deaths from diseases of blood: England and Wales 1960-1974

Cause of death	1960-64	1965-69	1970-74
All diseases of blood and blood forming organs ..	10376	10125	9289
Aplastic anaemia ..	1218	1399	1579
Agranulocytosis ..	128	123	120

If Dr Inman's suggestion that co-trimoxazole was causally related to over one-half of the fatal cases of agranulocytosis during the period of his survey was true, then one would expect a major increase in the frequency of this condition in the period since its introduction in 1968. No such increase was observed. Thus either the association between co-trimoxazole and agranulocytosis is a spurious one in that the latter caused the former rather than vice versa, or it is a real one but physicians do not suspect it and therefore do not diagnose it, being content to certify patients dying of co-trimoxazole-induced agranulocytosis as dying from the infection which accompanies the agranulocytosis. We submit that the case against co-trimoxazole is unproved as yet and that Dr Inman will have to produce much more impressive data before claiming that a causal relationship between this drug and agranulocytosis is established.

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¹ Office of Population Censuses and Surveys, *Mortality Statistics 1974 England and Wales*, OPCS Series DH1, No 1, table 6. London, HMSO, 1977

Anaesthetic waste gas scavenging systems

SIR,—We feel that the point that we made (21 May, p 1348) regarding the article by Dr R F Armstrong and others (9 April, p 941) has not been fully appreciated by the other correspondents on this subject. They have ignored the results quoted by Dr Armstrong and his colleagues who, for the five systems tested, showed definite superiority for passive systems when all systems were used with similar fresh gas flows on open circuits. The relevant figures extracted from the table

Atmospheric pollution with halothane, all readings when using open circuits

	Passive systems			Active systems	
	Enderby valve	Penlon-Papworth system	Coaxial circuit with Penlon valve	BOC with open circuit	Draeger with open circuit
Mean ($\pm$ SE) halothane level during session (ppm)	4.6 $\pm$ 0.34	3.4 $\pm$ 0.42	1.9 $\pm$ 0.14	4.0 $\pm$ 0.32	5.3 $\pm$ 0.41

in their article are shown in the accompanying table.

As the Draeger system under these circumstances had the worst performance we cannot understand the comment by Dr Armstrong and his colleagues (21 May, p 1348) in reply to our original letter that "only the active system supplied by Draeger was effective, the main reason being that this was the only system which did not leak significantly from areas other than the exhaust valve." Drs L Rendall-Baker and R A Milliken (p 1348) and Dr B M Wright (11 June, p 1535) argue on intuitive grounds that an active system must be the most efficient.

We agree that a good active system should be better than a good passive system but we hope that the misleading summary by Dr Armstrong and his colleagues does not lead to this particular paper being quoted as support for active systems when in fact the results given in the paper show that, when tested with similar circuits, passive systems were more effective than active systems.

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Litholapaxy

SIR,—Your leading article (9 July, p 79) on lithotripsy contains a repeated mistake in spelling of the word litholapaxy (Greek *λίθος* stone, and *λαπάξ*, evacuation). To spell it twice as lithalopaxy shows an ignorance of its derivation.

The Russian Urat 1 method, published in the USSR in about 1955, of electrohydraulic disintegration was shown by Yutkin in London nearly 10 years ago and tried out by a member of the Urological Club; it proved to be lengthy and uncertain. I mentioned it in an article on the history of lithotomy and lithotripsy in 1968.¹ Its use in 14 cases is well described by Mitchell and Kerr² in the account you quote; they stress the cost and the short life of the tip of the probe, but perhaps these can be overcome by realistic pricing and careful usage.

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¹ Riches, E W, *Annals of the Royal College of Surgeons of England*, 1968, 43, 185.

² Mitchell, M E, and Kerr, W S, jun, *Journal of Urology*, 1977, 117, 159.

Stress incontinence

SIR,—Your leading article on stress incontinence (2 July, p 2) makes no reference to exercise as one form of treatment that may be of value. Women who have suffered from difficult deliveries, chronic illness, malnutrition, exhaustion, old age, and so forth often have attacks of bladder instability.