However, it seems relevant to refer now to the incidence of vomiting in paracetamol poisoning.

In total, we have records of 392 patients who ingested analgesic overdoses which included paracetamol; 120 took paracetamol alone, 44 took Distalgesic (dextropropoxyphene and paracetamol) alone, 157 took paracetamol combined with other drugs, and 71 took Distalgesic combined with other drugs. Among these patients only 46 (11.7%) had vomited or were vomiting at the time of admission; and of the 63 who were treated with oral methionine, vomiting occurred in only 13. Further examination of these cases shows that all vomited before the treatment began but that this symptom did not persist during treatment.

Obviously further analysis of these data is required, but it is already apparent that the difference in the incidence of vomiting with this type of poisoning as reported from Edinburgh (77%)¹ and London (16%)² cannot be explained simply by the fact that many of the latter data are collected indirectly through the Poisons Information Service inquiries.

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¹ Prescott LF, Illingworth RN, Critchley JAJH, Proudfoot AT. *Br Med J* 1980;280:46-7.
² Vale JA, Meredith TM, Crome P, et al. *Br Med J* 1979;ii:1435.

Antenatal care and high-risk babies

SIR,—Regular antenatal assessment is important in the interests of fetal and maternal health. However, as your leading article "Quality not quantity in babies" (9 February, p 347) points out, the fall in British perinatal mortality has occurred before there has been a chance to introduce incentives for mothers to attend antenatal clinics. But the idea is being spread through the media—presumably because of the misinformed opinion of certain professional health workers—that failure of pregnant women to avail themselves of antenatal check-ups is a leading cause of death, illness, and subsequent handicap in newborn babies. The implication is that the mother is at fault.

About 70% of deaths in the first week occur in low-birthweight babies, and many—perhaps most—weigh less than 1.5 kg. In the light of present knowledge it is difficult to see how regular antenatal assessment might prevent this happening. Certainly most of the ill low-birthweight babies cared for in our own newborn intensive care unit are born to

women who book early at the antenatal clinic and receive regular and skilled antenatal care. They suffer from complications such as premature rupture of membranes, spontaneous premature onset of labour, hypertensive disease of pregnancy, antepartum bleeding, and fetal growth retardation. We know very little about the causes of these conditions and we know even less about their treatment. Regular antenatal supervision might lead to their prompt diagnosis—but then what? That there is still an ill or high-risk neonate to be cared for is a measure of the success of the obstetrician in preventing the death of the unborn baby. In many respects there is a positive correlation between the quantity and quality of antenatal care and the paediatric resources needed. The problem is that in many of our maternity hospitals technological advances in monitoring the health of the fetus have outstripped the availability of resources required to care for the resultant high-risk baby after birth.

Firstly, we must stop laying the blame for things we do not understand at the feet of our patients. Failure to do so merely magnifies the guilt that women are already burdened with when their baby dies so soon after birth. Secondly, if we want women to book early and attend the antenatal clinic so that they and their unborn babies may benefit from obstetric services, we must also ensure that delivery units and special care baby units have the proper staff and facilities to look after those same babies after birth. The message is simple—regular antenatal care won't make ill babies go away.

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Quality not quantity in babies

SIR,—In your leading article (9 February, p 347) you discuss an "amniocentesis programme to detect fetuses with lethal central nervous system abnormalities." I think I am right in saying that amniocentesis will detect open central nervous system lesions, but cannot distinguish between those of varying severity, let alone indicate which are lethal.

There is a similar widespread but mistaken belief that open myelomeningocele in the neonate is lethal unless operation is done. This is, of course, untrue—unless steps are taken to ensure that they all die—for example, by heavy sedation and inadequate feeding.

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Cephalopelvic disproportion and caesarean section

SIR,—Barbara Culliton and Mr Wallace Waterfall (8 December, p 1488) comment that the caesarean section rate in the United States has doubled over the last decade and I believe that many obstetricians are worried lest a similar trend occurs in this country. Dr Marion Hall (2 February, p 333) incriminates intra-partum fetal monitoring and refutes the explanation, given by these authors, that the rise is due to an increase in cephalopelvic disproportion.

While the incidence of absolute dispro-

portion may not have changed, perhaps the diagnosis of relative cephalopelvic disproportion has. The use of partograms highlights slow progress during labour,¹ while it has been reported that slow progress after dilatation of 7 cm may be associated with a difficult forceps delivery.² The clinician may now be more likely to anticipate such a delivery and, either in the best interests of the baby or for medicolegal reasons (17 March 1979, p 763), opt for caesarean section earlier than he would have done some years ago.

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¹ Philpott RH. *Br Med J* 1972;iv:163-5.

² Davidson AC, Weaver JB, Davies P, Pearson JF. *Br J Obstet Gynaecol* 1976;83:279-83.

Herpes zoster in pregnancy

SIR,—The letters by Drs Veronique Moens and K M Huntington (6 October, p 870) which followed Professor A P Waterson's interesting review of virus infections during pregnancy (8 September, p 566) suggest that zoster may be commoner than the 13 cases found by Brazin *et al* in their review of the literature.¹ However, in 33 years of detailed surveillance of zoster cases in a practice of 3800 patients, Dr R E Hope-Simpson (personal communication) has not seen a case in pregnancy, and none occurred in 87 cases of zoster reported from a Glasgow general practice.²

I wish to report three more cases of zoster in pregnancy. They have occurred within four months, during an epidemic of varicella, in a practice of 7800 with approximately 100 deliveries a year. None of the patients give a history of recent contact with varicella zoster virus, nor did they meet at antenatal clinics.

Case 1—A 25-year-old woman, gravida 2. Expected date of delivery assessed as 19 August 1979 by ultrasound scan, which gave a maturity of 19 weeks on 29 month. Rubella immune. Zoster rash appeared on 9 August (38 weeks' gestation), left T8 dermatome. She had a normal delivery on 25 August at the general practitioner maternity home, and returned to her own home four hours later. The baby appeared normal and healthy and has remained so. Complement-fixation test: 1/160 against varicella zoster virus on cord blood. The Epidemiology Research Laboratory, Colindale, was consulted antenatally and advised that zoster immune globulin was unnecessary. The patient had had a severe attack of varicella in childhood at an unknown age.

Case 2—A 31-year-old woman, gravida 3. Rubella immune. Last menstrual period 29 August 1979. Expected date of delivery 5 June. Zoster rash appeared 26 November (12 weeks' gestation) with right L1 distribution. Complement-fixation test: 1/320 for varicella zoster virus. Severe varicella at age 2 years 9 months. Pregnancy appears normal.

Case 3—A 25-year-old woman, primigravida. Rubella non-immune. Last menstrual period 7 October 1979. Expected date of delivery 14 July. Zoster rash on 26 November (seven week's gestation), left T10 dermatome. Complement-fixation test 1/320. The patient had severe varicella aged 5 years. The patient is booked for confinement in the specialist unit as she is a primigravida and 150 cm (4 ft 11 in) tall.

Zoster is less common in women aged under 30 years than in older people (6.4% and 9.6% of two series^{2,3}) but may be under-reported in pregnancy as it seems probable that the fetus is rarely infected.

The two normal cases reported to your journal by Drs Moens and Huntington, the 13 cases reviewed by Brazin *et al*,¹ and my first case total 16. Eleven babies were reported

normal and, of the remaining five, two had rubella infection (one stillborn) as a complicating factor. I shall report the outcome of the remaining two cases.

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¹ Brazin SA, Simlovitch JW, Johnson WT. *Obstet Gynecol* 1979;53:175.

² Ross CAC, et al. *J R Coll Gen Pract* 1975;25:29-32.

³ Hope-Simpson RE. *J R Coll Gen Pract* 1975;25:571-6.

Acute gingivitis as atypical infectious mononucleosis

SIR,—The report of two cases of atypical infectious mononucleosis in elderly people by Dr M W McKendrick and others (20 October, p 970) prompts me to describe a single case presenting symptoms which I think have not been previously recorded.

A 65-year-old man noted painful gingivitis affecting the interdental papilla between the first and second right upper incisors. The following day the gingivitis had spread to the greater part of the upper gum. Oral phenoxymethylpenicillin had no effect. On the third day a lymph node below the angle of the jaw was enlarged and tender on each side. A low fever (37.2°C) persisted for two weeks. Despite some malaise the man continued at work. On the fourth day a blood film showed atypical mononuclear cells characteristic of infectious mononucleosis. The total white cell count was $9.5 \times 10^9/l$. The Paul-Bunnell test was positive. The erythrocyte sedimentation rate was 25 mm in the first hour. Gingival swelling, tenderness, and a tendency to bleed persisted for two weeks from the onset, and swelling alone lasted for about two months. The lymph nodes were no longer tender after the first week, but they have remained enlarged and firm till now, three years later.

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Smoking and mortality of male doctors

SIR,—I read Mr P N Lee's paper (15 December, p 1538) with great interest. In the paper the author presented the observed and the expected number of deaths from nine causes had doctors shown the same improvement as men in social classes I and II as a whole, and came to the conclusion that in 1970-2 there were 34 fewer deaths among doctors which could be attributed to reduced cigarette smoking. However, if the observed number of deaths among doctors is compared

with the expected number of deaths among men in social classes I and II then one cannot help reaching the conclusions that (a) the number of deaths due to coronary heart disease and stroke has decreased considerably but that (b) the number of deaths due to stress-related diseases has increased.

There is no evidence to support the assumption that giving up smoking or reduction in the level of smoking results in increase in the stress-related diseases (on the contrary, the standardised mortality ratio (SMR) for stress-related diseases is lower for social classes I and II for the period 1970-2 than for 1949-53); so it is essential to estimate the number of deaths among doctors for the period 1970-2 if they had maintained the same level of smoking relative to social classes I and II. Hence the same SMR values for coronary heart disease (126), stroke (131), and lung cancer (61) will be used for the period 1970-2. It may be pointed out that to reduce or to give up smoking is a voluntary decision and therefore within the direct control of an individual. On the other hand, most of the factors affecting stress-related diseases are outside the control of an individual. So in order to make a realistic estimate for the deaths caused by stress-related diseases the SMR values of 1970-2 must be used.

From the accompanying table it is evident that by reducing smoking more effectively there were 56 fewer deaths among doctors. However, if the overall reduction in deaths among doctors due to all other factors is considered, on the basis of SMR values for the same period, then there is no significant difference in the overall mortality ratio (SMR₁ is 100.4 and SMR₂ is 100.4).

As more and more men belonging to social classes I and II are giving up smoking and caring more for their health these days and doctors, on the other hand, are to come under more stress due to the stringent financial cuts in the NHS, it is high time that doctors become aware of the dangers they are likely to face. It is imperative that they take effective steps to reduce their own stress without the help of drugs. Perhaps a bit of meditation and regular practice of yoga may be the simplest preventive therapy.

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SIR,—My analysis of the mortality of male doctors (15 December, p 1538) has provoked comments not only in your columns but also in other medical journals and in the lay media, some of which suggest that the emphasis of my conclusions may have been

Numbers of deaths and death rates from "stress-related diseases" in doctors aged under 65 over the period 1951-71

	Non-smokers	Ex-cigarette smokers	Continuing cigarette smokers
No of deaths	57	48	183
Death rate per 100 000 per year	64	60	100

misinterpreted; and Sir Richard Doll and Dr Richard Peto have suggested that I should clarify the points I was making.

My main conclusion was that "the benefits of giving up smoking may not be so great as has commonly been assumed," on the basis of the relatively small improvement in mortality in doctors aged under 65 compared with men in classes I and II over the period 1951-71 when a greater proportion of doctors than of men in classes I and II gave up smoking. A Adelstein (personal communication) has pointed out that classes I and II included Army officers in 1949-53 but not in 1970-72; but even after excluding them from the calculations doctors' overall mortality trends (standardised mortality ratio (SMR) changing from 89 to 81) were only about 2% better than the control group (SMR changing from 87 to 81). Approximate calculations based on the data of Doll and Peto¹ on the relative mortality of smokers, ex-smokers, and non-smokers suggest that if all the difference in mortality between these three groups were actually due to their smoking, one would expect doctors' mortality trends to be about 8% better than the control group. Even after allowing for the fact that Doll and Peto's estimates of smoking habits, based on only 67% of male doctors, may have over represented those smokers more likely to give up, the discrepancy appears to remain. When one further takes into account that for coronary heart disease, the disease contributing most to doctors' actual relative improvement in overall mortality compared with classes I and II, a similar relative improvement could also be seen over the period 1931-51, before doctors gave up smoking, the discrepancy becomes more marked.

Two explanations are possible for this discrepancy. One is that some of the differences seen in the overall mortality of smokers and ex-smokers is due to differences in factors other than their smoking habits. Evidence supporting this is given by Seltzer.² The second is that trends in factors other than smoking may have affected the mortality of doctors and the control group differently over the period considered. The relative contribution of each explanation to the discrepancy is open to doubt but the possibility that the first is important, especially in relation to coronary heart disease, should not be overlooked.

The second main point of my paper was to point out and discuss the fact that for certain diseases (suicide, cirrhosis of the liver, and accidents, poisonings, etc) which might be considered stress related the mortality trend of doctors was relatively worse than that of classes I and II over the period 1951-71. Several possible explanations were considered in my paper, one of which was that giving up smoking may have contributed in part to the worsening trend if smoking had acted to reduce stress. At first sight, the data in the table above may seem to rule out this hypothesis. However, the situation is complex. It seems basically more plausible that "stress causes smoking" than that "smoking causes stress." If this is the case and if it is only the less stressed smokers who feel able to give up, one may observe more stress-related disease in continuing smokers than in those who have given up even if giving up smoking is actually associated with increased stress.

Dr Lynn T Kozlowski and others (9 February, p 400) touch on some of the highly complex literature on smoking and stress. Though they quote evidence that smoking may directly reduce stress and that cigarette deprivation may induce stress, both consistent with the hypothesis described above, they suggest that in some cases smoking

The observed and expected number of deaths if doctors had maintained the same level of smoking relative to men in social classes I and II

Cause of death	1949-53 SMR ₁ *	1970-72 SMR ₂ *	1970-72		
			Observed	SMR	Expected
Coronary heart disease	126	97	261	126	340
Stroke	131	95	44	131	60
Lung cancer	61	48	31	61	39
Other malignant tumours	87	93	106	93	114
Bronchitis	48	60	13	60	22
Cirrhosis of liver	149	227	14	227	6
Suicide	192	356	55	356	15
Accidents, poisoning, etc.	88	175	60	175	34
All other causes	84	89	81	89	91
Total	101	100.4	665	92	721

*SMR₁ = SMR in doctors for 1949-53/SMR in classes I and II for 1949-53.
SMR₂ = SMR in doctors for 1970-72/SMR in classes I and II for 1970-72.