problems, and deafness,^{9,10} as well as sudden infant death.^{11,12}

The task of clearing the population of potentially dangerous viruses before immunisation may well prove impossible; but does that justify our setting up ill-founded contraindications, rather than real ones, and then shooting them down in order to prove that our vaccines are safe?

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SIR,—In answer to Professor R S Illingworth's letter (19 January, p 179) about the multitude of curious reasons given for not allowing children to be immunised against whooping cough, I would suggest the following.

Mothers have just undergone nine months of bodily change of varying discomfort followed by a less comfortable, and perhaps dangerous, process at the end of it, in most cases to produce (one hopes) a wanted child. Not only are they then asked to submit that child to a possibly damaging procedure (the risks of which have been quantified differently by different authorities) but they are asked at a time when they may be suffering from mild postnatal depression; and, as anyone who has ever felt depressed knows, this is not a time when one can confidently and rationally take a decision.

Sometimes fathers can help by giving a firm lead, but often the mother looks to the health visitor or doctor to decide for her—something which is debatably not justifiable in the circumstances. She is frequently happier if the immuniser misses out the whooping-cough vaccine and afterwards she may present as reason for this any unusual fact in the child's family or medical history. The medical adviser may consciously or unconsciously have considered this, but in fact he or she is attempting to respond correctly to the mother's doubts and fears, while also acting in the interests of the child to protect it by immunisation against other diseases—at the very least polio only, which involves no "jab."

I sometimes consider postnatal depression sufficient of an argument to start immunisation at six months, not earlier. This is consistent with immunology, although other factors may sway policy towards an earlier start.

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Mortality of male doctors and reductions in cigarette smoking

SIR,—We disagree with Mr P N Lee's analysis of the mortality of male doctors (15 December, p 1538) and his provocative conclusion that the

benefits of giving up smoking may not be so great as has commonly been assumed. His calculations, which are obtained by a complex manipulation of standardised mortality ratios, showed "savings" in deaths from three major cigarette related diseases: coronary heart disease (83 deaths saved), stroke (16), and lung cancer (8). These savings are compared with "losses" from three stress-related conditions: accidents, poisonings, etc (30), suicide (26), and cirrhosis of liver (4). The implication is made that the doctors have become more prone to these stressful conditions partly owing to a possible adverse effect of giving up smoking. The analysis is based on the Registrar General's occupational mortality tables for England and Wales, which restrict attention to men under 65 years of age.

Life certainly does not end at 65 years; in England and Wales the expectation of life for men at 65 is 12 years and the doctors can expect to live even longer. It is therefore of vital interest to have an overall analysis of "savings" and "losses" in deaths over the whole life span of the doctors. Unfortunately national data are not available for this task; the answers on the extent of benefits arising from giving up smoking for the particular group of British doctors lie with the follow-up study of Doll *et al.*^{1,2}

The national figures for male deaths attributed to these cigarette-related diseases (table) show that a large majority of deaths occur in the postretirement age group.³ In contrast, the number of deaths from the stress-related conditions thought to be partly attributable to giving up smoking largely occur in the working age group. These opposing trends clearly demonstrate that the "savings" would rise and the "losses" diminish in the postretirement age. In this context we cannot understand why Mr Lee believes that the effect of giving up smoking might be seen most clearly in the younger age groups. It is most unlikely that these trends observed in the general population would be markedly different in the doctors. Ninety per cent of lung cancer deaths occur among smokers.⁴ It is estimated that in England and Wales the number of lung cancer deaths that could be saved by giving up smoking (22 000 lives) far outnumber the total number of stress-related conditions (12 383) that could partly be attributable to giving up smoking. These estimates are based on the assumption that 50% of adult males smoke cigarettes.⁵

Sixty-three per cent of ischaemic heart disease deaths occur among smokers; the number of lives saved annually is expected to be over 23 000. We firmly believe that the number of lives saved by giving up smoking will far outweigh the lives lost by the adverse effect of stress-related conditions arising

from giving up if all age groups are included in the analysis of male doctors.

Mr Lee's restricted analysis up to retiring age showing a small gain of 5% in lives saved by the reduction of smoking in doctors is misleading and can seriously affect the preventive programmes on smoking. There might be a small percentage of smokers who would be better off smoking than giving up but it is for the smoker himself to weigh up the relative advantages and disadvantages. For the large majority of smokers, however, it is prudent to give up rather than to continue. We believe that this overall conclusion is true for every subgroup of the population, including the doctors.

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SIR,—The conclusion by Mr P N Lee (15 December, p 1584) that the tar reduction in cigarettes occurred before publication of the first tar table in 1973 and that the table was "on the industry's own initiative" deserves comment.

The first publication of tar and nicotine retrieval from cigarettes identified by brand was in 1967.¹ This article was published despite the opposition of tobacco interests, some governmental groups, and even several fellow scientists who deplored its practical message. Shortly after this publication and the subsequent listings by the Federal Trade Commission industry began to publish tar and nicotine levels as part of their advertisements.

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Prophylaxis against postoperative pulmonary embolism

SIR,—The comprehensive study by Dr U F Gruber and others (12 January, p 69) on the prevention of fatal pulmonary embolus will interest all pelvic surgeons. Two deaths followed 414 gynaecological operations (all in women over 40 years and lasting more than 30 minutes). One was after dextran and the

Number of deaths in smoking-related and stress-related conditions in two broad age groups for men (England and Wales 1977)

		Number of deaths	Age		Ratio of deaths
			<65 y	≥65 y	≥65 y <65 y
Cigarette-related conditions	Ischaemic heart disease	89 411	28 613	60 798	2.12
	Lung cancer	26 679	9 193	17 486	1.90
	Cerebrovascular disease	27 831	4 896	22 935	4.68
	Subtotal	143 921	42 702	101 219	2.37
Stress-related conditions	Accidents, poisonings, etc	9 029	6 691	2 338	0.35
	Suicide	2 363	1 851	512	0.28
	Cirrhosis of liver	991	640	351	0.55
	Subtotal	12 383	9 182	3 201	0.35

other after heparin prophylaxis. Both women were over 75 years and had malignant disease. This incidence (0.48%) of fatal pulmonary embolism is not experienced in Wolverhampton, where 17 139 gynaecological operations of all types have been performed during the past five years, of which approximately 36% were major pelvic procedures, a fifth of which was radical cancer surgery. No specific routine prophylaxis was used.

In the last five years only three patients died in the first four weeks after operation from pulmonary embolus (confirmed at necropsy), an incidence of 1 in 5713. One patient died from peritonitis and basal pulmonary thrombi following a small bowel resection for intestinal obstruction associated with pelvic metastases. One died from massive pulmonary embolism and advanced corporal carcinoma seven days after drainage of a pyometra. The third died from pulmonary embolus after laparotomy for an inoperable pelvic tumour, later found to be bladder carcinoma at necropsy.

Whereas a constant watch should be kept for venous thrombosis and pulmonary embolus, specific chemical prophylaxis, with all its known (and in this paper demonstrable) hazards, should be avoided in gynaecological patients. However, age greater than 65 years, gross obesity, severe lower limb varicosities, pelvic cancer, and a history of thromboembolism will lead to a consideration of specific pharmacological prophylaxis, especially if the patient is on the operating table for over 60 minutes. Otherwise post-operative patients with suggestive symptoms should immediately be considered for intravenous heparin therapy.

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Intravascular thrombosis during air travel

SIR,—I was very interested to read the case report by Mr R E C Collins and others (8 December, p 1478) on femoral artery thrombosis after prolonged travel. Initiation of intra-arterial thrombosis during travel by air, especially in older subjects, may not be so uncommon.

Recently, I saw an Asian woman of 76 who felt her right leg going colder than the rest of her body a few hours after take-off on board a scheduled flight from Karachi to London. On arrival, she did not experience ischaemic pains or sensory changes. Her right foot was cooler and arterial pulses below femoral were weaker on the right side than the left. There was an audible bruit over the femoral artery. During the following four months, there was no marked change in the state of her ischaemic limb. Arteriography or surgery was not performed.

The interesting features of this case are that the woman developed the ischaemic symptoms within a relatively short period of travel and that, apart from her age, there were no predisposing factors. She was in excellent health prior to her journey and had no history of pre-existing arterial disease. She had been normotensive, a non-smoker, and a teetotaler. Apart from the occasional anti-inflammatory agents for moderate osteoarthritis of her knees, she was not on any medication and had never taken any steroids. Having lived all her life in the tropics, she was accustomed to keeping herself well hydrated.

What proportion of all cases of intravascular thrombosis develop the pathology during or soon after air travel? Is flying in itself a factor? Sudden changes in pressure,

oxygen and carbon dioxide concentrations, and temperature occurring inside aircrafts on ascent and descent may possibly affect the haemodynamics. The effects of low temperature and oxygen lack on sympathetic activity are well known. Blood pressure may also be affected by the altered plasma levels of catecholamines due to temperature changes¹ or to the apprehension of flying. Furthermore, it has been shown that a sudden drop in atmospheric temperature is associated with the initiation of arterial thrombosis in the elderly.²

Air fares are cheaper and many more people travel by air now than ever before; and if flying, with or without other predisposing factors, does promote the formation of intravascular thrombus we should be aware of this hazard.

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Massive arterial thrombosis and oral contraception

SIR,—We were interested to see the case report "Massive arterial thrombosis and oral contraception" by Drs P M Holt and D Hollanders (5 January, p 19), and would like to make the following comments.

Firstly, the authors state that arterial thrombosis is less often encountered in association with oestrogen-containing contraceptives than abnormalities of coagulation or venous thromboembolic diseases and that "these events [arterial thromboses] are relatively rare." However, in the report of the Royal College of General Practitioners study¹ there were 10 deaths attributed to arterial thrombosis (seven myocardial infarction, one acute myocardial insufficiency, and two mesenteric artery thrombosis) compared with one death attributed to venous thromboembolism (pulmonary embolus) in women who had used oral contraception, either currently or in the past. Similarly, of the nine cardiovascular deaths reported in the Oxford-FPA study,² all of which were in women who had at some time used oral contraceptives, five were directly attributable to arterial thrombosis (three myocardial infarction, one chronic ischaemic heart disease, and one precerebral artery occlusion), while there was none due to venous thromboembolism. Thus although, as Drs Holt and Hollanders suggest, clinicians may still be relatively more aware of the association of oral contraceptives with deep venous thrombosis and pulmonary embolism, arterial thrombosis accounts for a much greater proportion of the increased mortality which may be attributed to oral contraception.

Secondly, although the occurrence of massive arterial thrombosis may be unusual, in a study of all deaths during 1978 in England and Wales in women aged 16-44 years we have identified three women dying of widespread large artery thrombosis without venous involvement, including the woman reported by Drs Holt and Hollanders. The accompanying table summarises the presence of risk factors in these three cases (the information being derived from coroners, hospital records, general practitioners, and family planning clinics).

As can be seen, only one patient was using oral contraceptives (Eugynon 30) at the time of death. Case 2 had stopped using oral contraceptives seven years previously and case 3 had never used them. The only characteristic which these women appear to share is that they were all moderate or heavy cigarette smokers at the time of their death.

Thirdly, anecdotal case reports play an important role in suggesting new aetiological hypotheses and in directing the focus of subsequent epidemiological research. However, they do not allow the estimation of relative and attributable risks. Nevertheless, Drs Holt and Hollanders do attribute risk to the current use of oral contraception—both implicitly in the title of their paper and explicitly in their penultimate sentence: "Massive arterial thrombosis . . . represents another major complication of oral contraception." We do not believe that there is sufficient evidence yet available to justify this statement. The data from our study indicate that cigarette smoking may play at least as important a role as the use of oral contraception in the mortality from generalised arterial thrombosis.

We would like to thank Drs J O Davies, A D Gordon, and J R Leslie for providing information and for giving us permission to report their patients; the coroners and medical records officers for their help with our study; and Dr J I Mann for his advice. The study is supported by the medical Research Council.

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Oral contraceptives and the prothrombin time

SIR,—Dr E De Teresa and others (17 November, p 1260) report that in 12 patients on long-term anticoagulation with warfarin the mean prothrombin time ratio was significantly higher, or the dose of oral anticoagulants required to get the same prothrombin time ratio was significantly lower, when they were also taking oral contraceptives. An opposite effect could be expected, on the assumption that some clotting factors tend to

Risk factors in three cases of massive arterial thrombosis

	Case 1 (reported by Holt and Hollanders)	Case 2	Case 3
Age	34	43	43
Smoking at death (cigarettes/day)	40	20	20
Hypertension	Present*	Absent	Absent
Nutritional state (from postmortem report)	"Very obese"	"Nutrition: good"	"Obese"
Hyperlipidaemia	Absent	Absent	Absent
Diabetes mellitus	No	No	No
Use of oral contraceptives	Current user	Ex-user	Never-user

*Blood pressure not recorded as abnormal until 11 months before death—then 125/85 mm Hg)