- All letters must be typed with double spacing and signed by all authors.
- No letter should be more than 400 words.
- For letters on scientific subjects we normally reserve our correspondence columns for those relating to issues discussed recently (within six weeks) in the *BMJ*.
- We do not routinely acknowledge letters. Please send a stamped addressed envelope if you would like an acknowledgment.
- Because we receive many more letters than we can publish we may shorten those we do print, particularly when we receive several on the same subject.

Heart disease in Asians

Drs Kim M Fox and Leonard M Shapiro have raised the question why the rates of ischaemic heart disease in Indians in Britain are higher than those of Europeans (30 July, p 311). The answer probably is diet.

The British diet is increasingly high in bran, dietary fibre, and fermented milk products such as yoghurt, which contain a high proportion of short chain fatty acids of the C_4C_6 series. Fibre ferments in the gut, liberating large quantities of acetic acid, the precursor of short chain fatty acids.¹ Thus a relative preponderance of short chain fatty acids in the plasma of Europeans is likely, even though their total lipid concentrations are similar to those in Indians living in the United Kingdom. On the other hand, the diets of Indians living in Britain have changed considerably from their traditional diets. Chapatis are made from refined wheat flour, which is low in fibre. Fermented milk products are hardly used. Legumes, dals, and lentils, which require much cooking with ghee and turmeric powder (*curcuma domestica*), have been replaced by fast foods. This is likely to result in a relative lowering of plasma short chain fatty acids. To my knowledge no study has been done of the chain length profiles not only of these two groups but even among Europeans with ischaemic heart disease versus healthy controls. Although speculative, such a possibility needs exploring because in experimental systems, while long chain fatty acids of not less than 16 carbon atoms enhanced thrombosis, short chain C_4C_6 fatty acids did not.^{2,3}

In the traditional Indian setting ischaemic heart disease rates are significantly lower than those of Europeans, but within India the disease is 15 times more common in railway sweepers from south India than in Punjabi sweepers in north India, who have similar physical activity at work, socio-economic state, psychological stress,⁴ and lipid concentrations.⁵ There are, however, striking differences in patterns of diet and eating, methods of cooking,^{6,8} and haematological factors. The mean clotting time is shorter among southern Indians than among northern Indians, with no differences in clot retraction, platelet numbers, and even platelet adhesiveness. Clot lysis is faster in northern Indians.³

The Punjabis, with a lower incidence of ischaemic heart disease, eat masticatory, fibre rich diets high in saturated fatty acids including ghee and fermented milk products such as yoghurt and yoghurt sherbet, whereas the high risk south Indians eat a non-masticatory diet of boiled rice, poor in fibre and 8-19 times lower in fats, all of which is based on polyunsaturated fats of gingili and groundnut vegetable oils (C_{18-21} , long chain fatty acids). The north Indians boil their fats with

turmeric powder, which breaks up the fat globules more than 200-fold and reduces their chain length, but the south Indians do not.^{6,8}

Short chain fatty acids get absorbed directly into the blood stream via the portal vein without chylomicron formation, whereas the long chain fatty acids get hydrolysed into simpler acids, for which bile is essential, and these in turn are resynthesised into complex long chain fatty acids in the wall of the intestine and are absorbed as chylomicrons via the lacteals (figure). In both

Pathways of lipid absorption. TG=triglycerides

population studies and feeding experiments the amount of bile in the intestinal lumen of south Indians was significantly higher than that in north Indians.³

Poole removed chylomicrons from blood by high speed centrifugation and showed that the clotting time of plasma was increased but returned to normal when the washed chylomicrons were returned to the plasma.⁹ Studies by several workers have shown no differences in atherosclerotic indices between south Indians and north Indians.

It would therefore not be unreasonable to suggest that changes in the traditional diet are perhaps the most important factors in the higher mortality ratio for ischaemic heart disease among Indians in Britain. A comparison of the chain length of plasma fatty acids and chylomicron profiles between these two groups and between Europeans with acute myocardial infarction and healthy controls would be illuminating. Finally, the higher incidence of diabetes and hypertension in the Indians living in the United Kingdom may be a confounding indirect association, rather than a causal association, because both these disorders are also probably diet dependent.^{10,11}

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Anti-D and miscarriages

May I add substance to Dr Marcela Contreras's reply to the excellent article by Dr C B Everett (17 September, p 732).

There is no doubt abortions are an important factor in Rh sensitisation. Data from the Yorkshire region support this (table I). During a five year period 22 mothers developed antibodies in the next pregnancy after a previous unprotected normal delivery and 16 after an unprotected abortion. In 1987, 5 out of 6 "new" cases followed an unprotected abortion. Dr Contreras quotes an analysis of 18 mothers sensitised after an unprotected abortion in 1980-3. More details of these cases are summarised in table II.

In this series after the four spontaneous abortions occurring at less than 10 weeks the antibody which developed in the next pregnancy was detected after 30 weeks and no infant required exchange transfusion. Even when the spontaneous abortion occurred at 10-20 weeks an almost similar pattern emerged except that one infant required exchange transfusion. The pattern was very different for termination of pregnancy.

These findings support Dr Contreras's view that spontaneous abortion before 14 weeks does not appear to be an important cause of maternal sensitisation, although one case (case 12) did require exchange transfusion. The outcome of terminations of pregnancy was a different story. Four of the nine infants in the subsequent pregnancies required exchange transfusion and one infant died.

Regrettably there are few data on threatened abortion. There is evidence of fetal red cell leak similar to complete abortion and most experts would advise that anti-D should be given. I am often asked how often anti-D should be given if a patient continues to bleed for weeks. My view is

TABLE 1—Incidence of maternal anti-D sensitisation and cause of new cases, Yorkshire region

	1983	1984	1985	1986	1987*
No of new cases	34	47	39	36	38
Causes for new cases:					
Primigravidae	6	9	7	7	7
Failure of administration of anti-D:					
After normal delivery	4	9	3	5	1
After abortion	5	Nil	2	4	5
Failure of protection	15	25	22	15	20
Not eligible	4	4	5	5	5
No of longstanding cases	62	54	52	52	51
Total	96	101	91	88	89

*Provisional.

TABLE 2—Failures of administration of anti-D after abortion in 1980-3 and outcome of subsequent pregnancy

		Subsequent pregnancy		
		Outcome		
Case No	Type of abortion	Antibodies first detected	Cord haemoglobin (g/l)	Exchange transfusion needed
<10 weeks' gestation				
1	Spontaneous	Delivery	145	No
2	Spontaneous	Delivery	165	No
3	Spontaneous	33 weeks	151	No
4	Spontaneous	32 weeks	165	No
5	Termination	18 weeks	100	1
10-20 weeks' gestation				
6	Spontaneous	Delivery	158	No
7	Spontaneous	Delivery	167	No
8	Spontaneous	Delivery	174	No
9	Spontaneous	30 weeks	141	4
10	Termination	35 weeks	Not done	No
11	Termination	36 weeks	171	No
12	Termination	32 weeks	114	No
13	Termination	27 weeks	133	4
14	Termination	18 weeks		Stillbirth, hydrops
15	Termination	16 weeks	117	1
Over 20 weeks' gestation				
16	Spontaneous	34 weeks	143	No
Gestation not known				
17	Termination	32 weeks	168	No
18	Termination	30 weeks	Not done	No

monthly. This corresponds as far as we can tell to red cell life and also to the length of measurable activity of the injected antibody.

There is no doubt that 250 IU (50 µg) anti-D immunoglobulin should be administered after all abortions, threatened or spontaneous, if they occur after 14 weeks and terminations at any time. In my view it is simpler to administer anti-D to mothers having a threatened or spontaneous abortion before 14 weeks, although I admit the chances of sensitisation are less.

More data are required to clarify this clinical conundrum. It may be possible to organise this on a national basis but, as Dr Everett states, much more clinical information will be required such as parity, length of gestation, and above all the type of abortion.

The Blood Products Laboratory issues roughly 50 000 ampoules of 250 IU a year. The only statistics available regarding abortions are so called legal abortions (presumably induced). In 1986 there were 172 000. If we assume that the incidence of abortions is not significantly different in Rh D negative and Rh D positive patients this is equivalent to 25 000 legal abortions in Rh D negative women. Thus about half of the 250 IU ampoules are used in legal abortions and the remainder for spontaneous, threatened, and induced abortions, plus amniocenteses, etc. Whether this reveals any gaps in prophylaxis I do not know.

Table 1 is also a reminder that the numbers of primigravidae developing antibodies and failures of protection in mothers receiving anti-D immunoglobulin show no sign of decreasing. Most of these cases are intrapregnancy sensitisations, thus unaffected by standard post-delivery anti-D injections. Antenatal prophylaxis is at present the

only measure available to us to prevent at least some of these cases.

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Monitoring during sedation for endoscopy

Dr Norman Tate (20-27 August, p 561) raised important points which need to be addressed not only by endoscopists but also by endoscopy assistants and, indeed, by others who perform diagnostic and therapeutic procedures without the help of anaesthetists on patients who are sedated. Although we are uncertain what he means by monitoring, few endoscopists or their assistants will accept his implied criticism that patients undergoing endoscopy are not competently monitored. In most patients electronic monitoring is unnecessary. All endoscopy is carried out by medically qualified staff who will have received training in cardiorespiratory resuscitation techniques. According to the recommendations of the British Society of Gastroenterology, two nurse assistants should be available for endoscopy lists, and a particular part of their training is that they should observe and care for patients at all stages during an endoscopic procedure. All endoscopy is carried out in hospital, and for this reason particularly Dr Tate's analogy with dental practice is a poor one. The endoscopist in hospital has rapid access to equipment and the resuscitation team, skill and facilities that may not be available to the dentist. Endoscopists are aware of the frailty of some of their patients, particularly those undergoing interventional biliary endoscopy, and take great pains to ensure adequate but safe sedation. Endoscopists regularly performing these procedures are at least as experienced as most anaesthetists in using benzodiazepines and opioid analgesics and in giving the care necessary for patients who are receiving such sedation. Agents for the reversal of sedation with these drugs are now available.

Despite these comments most endoscopists would happily accept the help of further staff to monitor patients during the procedure. Many units are desperately short of endoscopy assistants, and in the current financial climate it is impossible to obtain further staff.

Our policy is that endoscopists should be taught the considerate, safe, and efficacious use of sedation for endoscopic procedures by endoscopists and that monitoring of patients during the procedure should be the responsibility of all present, with one designated nurse assistant having the primary role of monitoring. Formal audit may not be widely published, but by adopting these guidelines we have encountered only three cardiorespiratory emergencies during interventional biliary endoscopy in the past five years in a unit that treats more than 400 new patients each year. All three affected patients were resuscitated promptly. The average age of our patients is 79 years; 60% are jaundiced, 20% have cholangitis, and over 40% have other significant medical disease. Many are

referred having been considered to be unfit for receiving anaesthesia elsewhere. We consider that these results testify to the safety of endoscopy even without the aid of cover by anaesthetists. We emphasise that we write not to criticise Dr Tate's valuable contribution but to ensure that this issue is considered in perspective, particularly by doctors who do not practise endoscopy.

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May I reassure Dr Norman Tate (20-27 August, p 561) that we physicians are indeed aware of the importance of monitoring the cardiorespiratory system during sedation for upper gastrointestinal endoscopy in patients who are old and frail. I have recently completed a study in which 50 consecutive elderly patients (mean age 73.2 years, range 65-89) undergoing elective gastroscopy were continuously monitored for arterial oxygen saturation by ear lobe pulse oximetry and for cardiac rhythm by electrocardiography. The patients were sedated with intravenous diazepam 0.1 mg/kg body weight. In all the patients arterial oxygen desaturation occurred to some extent during the procedure (mean fall in percentage arterial oxygen saturation 5.7, range 1-14), but none suffered clinically important arrhythmias. Bell *et al* observed a similar trend in arterial oxygen saturation during gastroscopy in a larger, but younger group of patients sedated with midazolam, though they did not record the electrocardiograph.¹ Furthermore, they showed that desaturation could be abolished by the concurrent administration of oxygen through nasal cannulae.²

These studies confirm that fiberoptic upper gastrointestinal endoscopy performed on patients lightly sedated with a benzodiazepine is a safe procedure. Patients for whom there is concern about their respiratory state may be given nasal oxygen to prevent desaturation.

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Fish oil and plasma fibrinogen

Dr Arne T Høstmark and colleagues (16 July, p 180) suggest that one of the mechanisms responsible for the beneficial cardiovascular effects of fish oil may be the lowering of the plasma fibrinogen concentration. After giving about 6 g ω-3 fatty acid for six weeks to healthy men they noted a—maximal—13.2% decrease in the plasma fibrinogen concentration.

In a comparable study in 33 patients without diabetes and with stable claudication we could not discern such an effect. In a double blind trial one group (n=16) received six capsules of ω-3 fatty acids daily, with a total amount of 1.8 g eicosapentaenoic acid (C20:5, ω-3) and 1.2 g docosahexaenoic acid (C22:6, ω-3); the other group (n=17) received six capsules of corn oil daily containing 3 g linoleic acid (C18:2, ω-6). Plasma fatty acid concentrations were determined as a measure of compliance. Fibrinogen was measured with a Precimat fibrinogen kit No 524484 (Boehringer Mannheim Diagnostica, Mannheim, Federal Republic of Germany). Mean plasma