make a telephone call as part of the consultative relationship. The family doctors are rapidly accessible and value the personal contact so highly that they take almost all such calls immediately. They are of course usually in their office practice particularly in the afternoons because both they and most of the consultants have evolved the habit of doing hospital and other such work in the mornings, so that both parties do their "office" work in the afternoons. Moreover, I find that I write a better letter (that is, more useful to the referring doctor) after discussing the problem and its management with him.

The information available on first seeing the patients varies from (alas, all too often) virtually none to a detailed account of the problem on which my opinion is being sought. Often in the case of patients coming from a distance ("out of town") the referring doctor will telephone before referring the patient and this will expedite matters—for example, by leading to our agreement that to save the patient time he had better be admitted rather than try to complete the relevant investigations as an outpatient. As with most specialists the usual consultation requires two visits, one to assess the problem and the second to review the findings on investigation and "wrap up the consultation" with the patient. It is my usual practice to phone the referring doctor after the first visit to discuss what I have found and plan to do and then the definitive letter after the second visit can usually be prepared without a further telephone call. However, very commonly, particularly with complicated problems in which the history or the attitude and understanding of the patient is particularly important, I find it valuable to try to have all three parties "present" and then I will call the referring doctor while the patient is still with me and both he and the referring doctor hear my every word.

A further advantage is in deciding who does what. Shall I hang on to the patient until I resolve the problem (for example, the asthmatic whose drug regimen may take several visits to sort out) or may I see the patient for follow-up and if so at what intervals?

From what I have said it should be clear that this personal contact develops and grows one's relationships with the referring doctors and leads both parties to recognise each other's style. I could go on and on but will close with a final commendation from the standpoint of the teacher. I am sure the resident profits greatly as a trainee consultant by hearing these telephone consultations and even participating in them on a telephone extension. He then writes a better letter on my behalf—and to a doctor who knows that I am responsible.

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Paracetamol poisoning

SIR,—Mr J G Harvey and Dr J B Spooner (16 September, p 832) suggest that "the recorded number of paracetamol deaths considerably exceeds the actual number which can be directly attributed to paracetamol." The following account of 14 such cases reported to coroners in the north-east of England in 1974-6 further supports such view.

During 1974-6 six patients referred to the North-east Regional Paracetamol Overdose Service died in acute hepatic failure

complicated by severe disturbance of the clotting mechanism and a variable degree of renal dysfunction. The paracetamol overdose was in the region of 50 g or over. Indeterminate amounts of other non-hepatotoxic drugs and small quantities of alcohol were also taken. Only one of these patients was admitted early enough for the treatment with cysteamine (8 h after the overdose). Necropsy revealed "massive" hepatic necrosis and acute renal tubular necrosis of varying severity in all six patients. The majority of them also sustained major gastrointestinal bleeding and showed extensive petechial haemorrhage in the gastric and small-intestinal mucosa, with true superficial erosion in two cases. The prothrombin time ratio was severely altered before death and was in a range of 9-21%. In no instance could death be attributed solely to acute renal failure.

Further study of post-mortem protocols, coroners' files, and forensic laboratory records disclosed an additional eight cases of death in the region which had been reported to coroners as due to paracetamol overdose. When all the available data were scrutinised I came to the conclusion that only one of these patients died of pure paracetamol overdose. His death occurred in hospital four days after ingestion of the drug and was due to acute hepatic failure.

Of these eight cases of "sudden death" reported to coroners, one occurred within a few hours of alleged paracetamol overdose but on post-mortem examination it was shown to have been due to aspiration of gastric contents; a high blood alcohol level was detected. Six patients were found dead at home and empty bottles with missing known amounts of tablets were discovered by relatives or police by their bedside. Two of these patients had ingested large amounts of Distalgesic (dextropropoxyphene and paracetamol), one had swallowed a large, toxic dose of Lobak (chlormezanone and paracetamol), and in the remaining three cases only therapeutic blood levels of paracetamol were detected which could not have contributed to death. The findings in all 14 apparent cases of fatal paracetamol self-poisoning are summarised in the table.

Role of paracetamol as a cause of death

Drugs ingested	Interval between overdose and death	No of patients
Paracetamol alone	4-5 days	2
Paracetamol + small amounts of alcohol and non-hepatotoxic drugs	3-6 days	5
Paracetamol + large amount of alcohol	?	1
Distalgesic, Lobak	(found dead)	3
	(found dead)	3
Unrelated mode of death + paracetamol in therapeutic dose	(found dead)	3

Since the first report of two fatal cases of paracetamol overdose in 1966 by Davidson and Eastham¹ it has become widely accepted that a large overdose of paracetamol may cause subtotal hepatic necrosis leading to death in acute hepatic failure at least several days after the overdose. In Clark's series² 12 deaths after paracetamol overdose occurred 4-18 days after ingestion of the tablets. Only one case of fatal renal tubular necrosis in the absence of hepatic failure was recorded by Prescott³ in 1971. Since paracetamol is a common ingredient of many analgesics which may have a depressive effect on the central nervous

system (for example, Distalgesic), if an overdose of any of these is taken death may occur after several hours rather than days and may be erroneously attributed to paracetamol rather than to the other ingredient.

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¹ Davidson, D G D, and Eastham, W N, *British Medical Journal*, 1966, **2**, 497.

² Clark, R, *et al*, *Lancet*, 1973, **1**, 66.

³ Prescott, L F, *et al*, *Lancet*, 1971, **1**, 519.

Changing social-class distribution of heart disease

SIR,—In his letter (18 November, p 1431) Professor T R E Pilkington questions the explanation by Dr M G Marmot and others (21 October, p 1109) of the excessive rise in coronary heart disease after 1951 among men in social classes IV and V. He says that a more likely explanation is to be found in that "a large number of classes IV and V in the younger age groups can be expected to be immigrants from overseas and Ireland."

Although the proportion of immigrants is higher among men in social classes IV and V at ages 35-44 (18% as opposed to 13% for all men), mortality rates are similar to those of men born in England and Wales. As the table shows, exclusion of immigrants—that is, persons born outside England and Wales—does not make a difference.

Hypertensive disease and ischaemic heart disease (ICD 400-414): deaths/million/year in men aged 35-44

Social class	All countries of birth	Excluding those born outside England and Wales
I and II	525	513
IV and V	877	884
Ratio I and II IV and V	0.598	0.58

Mortality of immigrants by social class is discussed in the latest decennial supplement.¹

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¹ Office of Population Censuses and Surveys, *Occupational Mortality 1970-72*, Series DS No 1, pp 184-8, London, HMSO, 1978.

Selection of patients for dialysis and transplantation

SIR,—With reference to your leading article (25 November, p 1449) I too have difficulty in reconciling the epidemiological aspects of terminal renal failure—40 patients per million per annum—with the fact that only 16 patients per million per annum receive treatment in Britain.

There seem to be two possible explanations: either the epidemiology is incorrect or criteria for exclusion from dialysis are too harsh. The prevalence of chronic renal failure in the British population is not dissimilar to that in Europe, so presumably the epidemiological