excellent, and I am sure that they acted with perfect propriety in what must have been an embarrassing situation for them. When journalists asked me to identify the programme involved I refused to do so, because I wished to protect Mr Deehan's staff. He has had no such qualms about trying to discredit me.

Attention must now be focused on the second *Panorama* programme. This has been hailed as a concession to the doctors ("Panorama critics win rehearing"; "BBC backs down over transplants"; "As Mr Jenkins told the Commons this would give the medical profession a chance to refute effectively the charges made in the original programme.") It was even reported that the *Panorama* team would this time consult fully with the (British) medical profession. In preparation for this a number of experts have been discussing how best to explain to a television audience what brain death is, how it is diagnosed, and why there seems to be such controversy about the role of the EEG.

It now appears, however, that the BBC sees this programme in quite a different light. Replying to a letter from a prominent lay person, who expressed satisfaction that there was to be another programme to put the record straight, the editor of *Panorama* wrote (6 November): "I don't believe that our record is in any need of the correction you suggest. We are doing a second programme not because we thought the first one wrong, but because of the immense interest in this subject both by doctors and by the general public." In view of this attitude, and of the experience of the first programme, the experts in the field are unwilling to appear on the second programme unless the BBC agrees to certain conditions about the format and content, which have been formulated by the royal colleges and the BMA. Should the BBC not agree to these conditions but none the less transmits a second programme, it should be recognised that this will not include the representative views from British experts.

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Diagnosis of brain death

SIR,—I wish to address myself to the problem of the diagnosis of brain death. These comments are specifically directed to the British code on brain death, the BBC television programme presented on 13 October ("Transplants: Are the Donors Really Dead?"), and the variety of responses from the British medical community.

I believe that the diagnosis of brain death could be made with a high degree of certainty if the British code was followed exactly to the letter, with absolutely no error in clinical judgment. But to quote Hippocrates, "Life is short, the art is long, the occasion fleeting, experience fallacious, and judgment difficult." We are dealing with a situation where it is medically acceptable to diagnose a dead brain as alive, but it is never justified to diagnose a live brain as dead. Therefore we are committed to use a set of criteria that should not permit even minuscule uncertainty in the diagnosis of brain death. There should be no uncertainty in the minds of the physicians pronouncing brain death, or of those involved in the transplant procedure, and no uncertainty in the public domain—including potential donors. It should be made unmistakably clear that the

classical criteria of death (irreversible absence of heart beat, respiration, and movement) and the criteria for brain death in situations where the patient is being artificially maintained by life support systems represent different criteria for the pronouncement of death. We are not dealing with two different kinds of death but different criteria for diagnosis.

Since there are inherent errors in all scientific observations, it is incumbent upon us to use all those measures that are practically available. The British code demands that the diagnosis should not be in doubt and there should be no evidence of drug intoxication, and indicates that repeated examinations may be performed for periods as long as 24 hours. There are situations in which the primary diagnosis may not seem to be in doubt, but if one is dealing with multiple aetiologies, such as drug intoxication and intracranial haemorrhage, for example, it may be difficult if not impossible to absolutely rule out the contribution of the drug to the patient's condition. In other words, there is a possibility of error.

Since intoxicants can mimic the clinical findings of "brain stem death" for prolonged periods, the additional ancillary tests for electroencephalographic activity and cerebral blood flow are required to decrease the possibility of a misdiagnosis. Therefore, depending on the circumstances, the more stringent the criteria the less probable the error in diagnosis.

Although repetition of the evaluation might be as long as 24 hours according to the British code and the major purpose in the diagnosis of brain death is transplantation, examinations could be made less frequently. This also leads to a potential error. In our part of the "collaborative study," criteria for brain death were primarily studied independently of transplant requirements. I mentioned 14 patients who on initial examination were comatose, apnoeic, and cerebrally unresponsive, with absent cephalic reflexes, who subsequently survived. In 12 drug intoxication was the aetiology. In the remaining two patients the aetiology was not intoxication but transient cardiac arrest in one, and probable brain stem vascular insufficiency in the other. Both these patients had electroencephalographic (EEG) activity at the time of examination and when re-examined within 12 hours showed return of cephalic reflexes and respiratory activity. They were discharged subsequently in a neurologically normal state. Strict adherence to the British code in conjunction with appropriate clinical judgment might not result in diagnosing these patients as brain dead, but what if they were examined less frequently or if their condition had persisted for over 24 hours?

I believe that the criteria for diagnosis of brain death should be considered as an issue independent of the problems related to organ transplantation. The British code, however, links the diagnosis of brain death irretrievably with transplantation. If this position is maintained, should not the criteria be as strict as Dr Nesbakken suggests and include not only EEG but even studies of cerebral blood flow? This would obviate any questions of uncertainty and be applicable to the problem of brain death and transplantation in children and infants as well. It is in this group, especially in younger children, that the diagnostic criteria have been found to be less reliable.^{1 2}

Our ability to deal with the enormous complexities of the process of the death of an

individual is essential given the ever-increasing requirements for organ transplants.³

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¹ Ashwal S, Schneider S. *Ann Neurol* 1979;6:512-7.

² Korein J, ed. *Ann N York Acad Sci* 1978;315:1-454.

³ Korein J. In: Cotrell JE, Turndorf H, eds. *Anesthesia and Neurosurgery*. St Louis: CV Mosby Co, 1980: 282-321.

SIR,—The article by Dr P W de Leeuw and Professor W H Birkenhäger (1 November, p 1181) describing hypothermia occurring in an inpatient treated with prazosin highlights yet again the problems which may occur with a variety of drugs. In the home environment the elderly may easily become hypothermic when subjected to relatively small temperature drops, while an increasing problem is the younger sportsman—canoeist, sailor, pot-holer, mountaineer—subjected in an emergency to sudden temperature drops—for example, in the Fastnet disaster. I can record from personal experience frostbite affecting two toes together with symptoms of generalised hypothermia, under conditions where this would not normally be expected, in an individual taking propranolol. I would be most interested to hear from any practitioner who has similar data occurring in any situation.

I also read with interest the letter from Mr Robert Sells (1 November, p 1212) in which he notes that—for donor purposes—"re-examination of the corpse at a temperature of more than 35°C was, of course, mandatory." At a recent symposium at Yosemite, California, Dr Cameron Bangs was emphasising (with reference to accidental hypothermia) that "they are not dead until they are warm and dead." Surely all victims found apparently cold and dead, whether prospective donors or not, should be given the "mandatory" rewarming to 35°C before the all-important "final" decision is made? This is certainly not being done at present, so how many lives are lost?

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Millions of mild hypertensives

SIR,—Your leading article "Millions of mild hypertensives" (18 October, p 1024) encourages general practitioners to treat middle-aged persons in Britain with diastolic pressures over 100 mm Hg. The article goes further and points out that it may prove necessary to treat diastolic pressures over 90 mm Hg. We agree that this advice may prove correct but question whether there are actually four million such persons in Britain. The high prevalence figures are presumably derived from surveys of casual blood pressure, and, although casual blood pressure is important in predicting subsequent mortality, the general practitioner can be expected to treat hypertension only when it is sustained. When a patient's blood pressure falls to "normal" levels owing to familiarisation with the measurement or the surroundings or to natural fluctuation of the condition, the general practitioner will not start treatment. There are 15 million persons aged 40-64 in Britain and it would appear unlikely that four million of these have a sustained diastolic pressure equal to or over 90 mm Hg. The proportion with sustained

diastolic hypertension may be expected to be one-third to a half of those with a casual elevation of pressure^{1,2}—that is, less than two million.

We cannot condone the indiscriminate labelling of persons as hypertensive when they have only a transient anxiety-related rise in blood pressure. These patients, if treated, may experience a marked reduction in blood pressure and such treatment may not be in the best interests of the patients. As Anderson has shown,³ from the Framingham data, cardiovascular events do not increase until the diastolic blood pressure exceeds 90-99 mm Hg. There may be no benefit from reducing lower levels of diastolic pressure.

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¹ Hawthorne VM, Greaves DA, Beevers DG. *Br Med J* 1974;iii:600-3.

² Finnerty FA, Shaw LW, Himmelsbach MD. *Circulation* 1973;47:76.

³ Anderson TW. *Lancet* 1978;ii:1139.

The coronary care controversy

SIR,—We are grateful to Drs J M Rawles and A C F Kenmure (20 September, p 783) for drawing attention to two aspects of our randomised home-versus-hospital study in suspected myocardial infarction which merit fuller description.

The first is deaths before the arrival of the team. In our trial we dispatched a vehicle, with resuscitation equipment, a doctor, and a nurse, to patients who were registered with participating general practitioners and in whom a myocardial infarction was suspected. Inevitably, calls for help were also received from other sources and for conditions other than infarction and our paper reported on the first 500 calls which we had received, irrespective of their source. Of the 14 deaths before the arrival of the team, nine related to calls for help which had not come from participating doctors but from ambulance control, who asked us to dispatch equipment and a doctor to some special emergency (two children found unconscious at the bottom of a swimming pool; a miner who collapsed at the coal face; four patients found collapsed by neighbours or relatives; two patients who collapsed while non-participating GPs were with them and in whom external cardiac massage was already being performed when the team arrived but who remained in asystole). The remaining five deaths occurred in patients referred to us by participating doctors and are therefore the ones which directly relate to our study:

- (1) Time between onset of symptoms and call to GP (patient delay) 90 minutes; from this call to GP's call for team (GP delay) 15 minutes; from call to team to their arrival (team delay) 35 minutes. Patient dead when team arrived.
- (2) Patient delay 28 hours; GP delay 110 minutes; team delay 20 minutes. Patient thought to have been dead for some time when team arrived.
- (3) Randomised to home group on day before death. GP paid routine visit but was recalled five hours later because of sudden collapse and asked team to visit. Patient dead when team arrived 17 minutes later. Necropsy showed acute haemorrhagic pancreatitis and no myocardial infarction.
- (4) Patient presented with one-week history of vague chest pain and was sent home from GP's surgery. GP asked team to go to house but on their arrival 30 minutes later the patient was dead.
- (5) Patient delay 110 minutes; GP delay 15 minutes; team delay 23 minutes. On arrival GP

was performing external cardiac massage but heart could not be restarted.

Thus nine of the deaths reflect the pattern of emergencies in our area during the study period; and five, which occurred in participating patients with suspected infarction, illustrate the problem of delay caused by patients and we are completing a study of ways of reducing this.

The second aspect we should like to amplify is our at-risk population. Drs Rawle and Kenmure assume that all our general practices began referring patients to us from the outset of the four-year study period. Recruitment of each participating practice, however, required many briefing visits; so recruitment was serial and only the two largest practices were involved in the study for the whole four years. Their conclusion that we randomised "a mere 12% of the estimated number of coronaries" is thus invalid. Moreover, although it is necessary to remember this relationship between our randomised patients and the total infarct population if one wishes to apply our study to other communities, it does not invalidate the conclusions which we drew from the randomised patients themselves—namely, that for the majority of patients to whom a general practitioner had been called because of suspected infarction subsequent hospital admission conferred no advantage.

There are many considerations to take into account in planning coronary care for a community and our subsequent studies will, we hope, furnish useful information on our ability to shorten patients' delays by an intensive advertising and educational campaign and on the value of restructuring the ambulance services to take account of the emerging Association of Emergency Medical Technicians. The results of these studies should add to a growing body of information which will enable the debate about community coronary care to be conducted within a rational rather than an emotional frame of reference.

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Heart attack, stroke, diabetes, and hypertension in West Indians, Asians, and whites

SIR,—The findings of Dr J K Cruickshank and others (25 October, p 1108) that heart attack rates in West Indian immigrants are lower than expected is not surprising when one considers they have spent the earlier part of their life in a country where myocardial infarction and its risk factors are uncommon. The exceptional factor is hypertension, which when considered alone does not appear to be a risk factor in the aetiology of myocardial infarction in Jamaica.¹

My own experience while working in a semi-rural community in Ife, Nigeria, would support this. In a one-year period (1977) the new cases of hypertension, stroke, diabetes mellitus, and heart attack seen by the medical department of one small hospital were: hypertension 84, stroke 27, diabetes 32, myocardial infarction nil. As in the Jamaican study, the influence of affluence and Western diet, smoking, and sedentary occupation were low. Hypertension accounted for over 50% of cardiovascular disease, and was a considerable cause of mortality, usually from cerebral haemorrhage, hypertensive heart failure, or renal failure. Despite the high prevalence of diabetes, no case of myocardial infarction was seen over a three-year period.

In a Western environment people smoke more and eat more, especially in the form of "junk" foods, and they are certainly more sedentary. Thus in black immigrants we now

see patients with myocardial infarction in the UK (albeit a lower incidence) and, of course, there are reports of myocardial infarction from the more affluent patients in the urban areas of countries such as Jamaica and Nigeria. Will there still be a difference in second-generation blacks born in this country? If so, then we can start wondering more about the role of high blood pressure. To explain the difference between Asian and West Indian immigrants one needs to examine more closely the differences in other risk factors.

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¹ Ashcroft MT, Desai P. *Lancet* 1978;ii:1167-70.

Popliteal cyst rupture in normal knee joints

SIR,—The paper by Drs D G Macfarlane and P A Bacon (1 November, p 1203) is a welcome addition to the literature on the painful swollen calf. In the last three months I have seen three ruptured Baker's cysts in the course of general medical admissions. Only one was associated with rheumatoid arthritis, the other two occurring in fit young men.

A 28-year-old civil servant had experienced intermittent swelling of the left knee joint over the two years following a cartilage extraction, the episodes being related to playing football. Occasionally a swelling at the back of the knee joint was present simultaneously. Over the week before admission such a swelling had been present, but it had disappeared on the day before admission in synchrony with the onset of paraesthesia in the left calf. Clinical examination showed there to be a red, tender, swollen calf and an effusion in the left knee joint. Pitting oedema was present over the shin and calf but there was no pedal oedema. Symptoms and signs settled rapidly with rest.

A 26-year-old scaffolder fell nearly 4 metres and landed on a straightened left leg two weeks before admission. The leg was painful but he continued to work normally. He was awoken by a tight discomfort in the left calf. On the following day he was found to have a red, tender, swollen left calf associated with a large knee effusion, a fullness in the popliteal fossa, and pitting oedema over the lower leg but not over the foot. Conservative treatment brought about a resolution of the inflammation within 24 hours.

In the management of a painful swollen calf a full history and careful examination of the whole of both legs are essential.¹ It has been suggested that ruptured Baker's cysts are clinically indistinguishable from calf deep-vein thromboses.² In the two cases reported here definitive pitting oedema was present over the shin and calf but absent over the foot; in the case of the rheumatoid patient pitting oedema was disproportionately severe over the lower leg compared with the foot. Deep-vein thrombosis of the calf is often associated with pedal oedema but rarely is pitting oedema seen over the shin but not the foot. I venture to suggest that this clinical sign may provide an additional aid to the diagnosis of ruptured Baker's cyst.

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¹ Hughes GR, Pridie RB. *Proc R Soc Med* 1970;63:587-90.

² Simpson FG, Robinson PJ, Bark M, Losowsky MS. *Lancet* 1980;ii:331-3.

SIR,—The report by Drs D G Macfarlane and P A Bacon (1 November, p 1203) fails