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

to mention the possibility that joint rupture could be the presenting feature of inflammatory arthritis. When joint rupture complicates arthritis it is almost always restricted to early joint involvement.[1] In patients presenting with joint rupture systemic examination and the investigation should be performed to exclude this possibility. Even if no obvious cause is found at the time of presentation many patients will develop frank local synovitis or evidence of a generalised rheumatic disease within a year or two.

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SIR,—We were interested to read the report by Drs D G Macfarlane and P A Bacon of popliteal cyst rupture in normal knee joints (1 November, p 1203). This finding is, however, not entirely new. One of Baker's original eight cases[1] had no evidence of disease of the knee joint until the joint became infected after aspiration of the cyst and one other had only slight swelling of the knee since a sprain two years previously. Baker also gives the history of an army officer (originally described by Foucher in 1856) who developed a chronic knee effusion after a forced march on rough ground and who subsequently developed a popliteal cyst which ruptured. His symptoms eventually resolved and left him with an apparently normal knee.

In our own series of 43 patients presenting with symptoms suggestive of deep vein thrombosis,[2] 16 had popliteal cysts, of whom only two had rheumatoid arthritis, six had mild degenerative joint disease, and eight had no previous symptoms referable to the knee, though one of these had crepitus on passive flexion. Furthermore, five of these 16 patients had venographically proved deep vein thrombosis as well as popliteal cysts. We suggest that the presence or suspicion of popliteal cyst should not lead the clinician to conclude that a deep vein thrombosis can be excluded.

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Toxic shock and tampons

SIR,—The leading article "Toxic shock and tampons" (1 November, p 1161) stated that the syndrome is as yet unreported in Britain.

I suffered the characteristic symptoms on three separate occasions in 1978. On the first occasion I had just been fitted with a contraceptive diaphragm and was somewhat inexpert at inserting it. On the day following the first occasion of its use I developed, in a matter of hours, high fever with rigors, a centripetal erythematous rash, vomiting, diarrhoea, muscle tenderness and stiffness, backache, and severe pelvic pain. This lasted about four days and the symptoms subsided quite abruptly, leaving a residual pain in the left iliac fossa. On this occasion my GP prescribed co-trimoxazole, believing the illness to be an atypically presenting salpingitis. The second episode was 20 days after the first and again followed insertion of the diaphragm. The symptoms were the same but in addition I suffered severe vaginitis and a purulent vaginal discharge. Cultures from the swabs taken showed *Staphylococcus aureus*. The third episode occurred 16 days after the second. This time I was on holiday, menstruating and using extra-absorbent tampons (Lil-lets Super Plus). I experienced the same symptoms but they lasted barely two days.

By this time I was extremely worried and on my return home I consulted a gynaecologist, who could find no other explanation than salpingitis for my symptoms (and the continuous left iliac pain). Since then the pain has gradually disappeared; I have avoided using internal tampons (especially the highly absorbent kind) so far as possible, and greater skill in inserting the diaphragm has meant less possibility of internal trauma. On reading the reports of "toxic shock syndrome" this year,[1] I immediately recognised the cardinal signs of my mystery illness of 1978.

I think that it is important to consider that tampons need not be the only cause of "toxic shock syndrome" and suggest that inexpert use of the contraceptive diaphragm may also give rise to the condition, which can occur therefore at times other than during menstruation.

MEDICAL STUDENT

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SIR,—We read your leading article on toxic shock and tampons (1 November, p 1161) and agree with your statement that "it would be curious indeed if such a disease were to be confined to the United States." The syndrome has been described in Sweden,[1-2] and we want to report a further case with severe circulatory symptoms.

The patient was a 19-year-old woman with normal periods who used tampons of a common Swedish type. She fell ill on the fourth day of her period, with pains in her body and joints, severe headache, and high fever, followed by severe vomiting and watery diarrhoea. Because of rapid deterioration she was brought to hospital 36 hours after the onset of her illness. On admission she was in shock; her blood pressure was 70/55 mm Hg; she had a tachycardia with a pulse rate of 150 beats/min; and her temperature was 40·6°C. Bilateral conjunctivitis was noted but there were no exanthema.

The patient had a foul-smelling discharge from the vagina, and *Staphylococcus aureus* of phage group I and *Escherichia coli* were grown from vaginal secretions taken from the tampon. The patient was treated with intravenous fluids, plasma, methylprednisolone and cefuroxime. An improvement in central as well as peripheral circulation occurred, urine production started, and the central venous pressure rose to 8·5 cm water within three hours of treatment. After 12 hours of treatment, however, the blood pressure as well as the central venous pressure fell, diuresis stopped, and the patient became disoriented. Dopamine hydrochloride and digitalis were added to the previous treatment and produced a prompt improvement in her circulation and diuresis. Two days later, however, her heart was dilated, and she had symptoms of pulmonary stasis, but no pericardial effusion could be seen. An extreme sinus arrhythmia with pulse rates down to 35 beats/min was noticed. The decompensation was treated symptomatically, and the symptoms slowly disappeared. A week after starting treatment all signs of cardiovascular problems had disappeared.

Cultures from the blood, cerebrospinal fluid, and urine were negative at admission. *Staph aureus* of phage group I was cultured from the nasopharynx as well as from the tampon. On the third day the patient had a typical strawberry tongue, and within a fortnight intense desquamation of the palms occurred. But she never developed any exanthema during the course of the disease.

This patient fulfils the criteria for the toxic shock syndrome (as reported by Daley *et al* and Davis *et al* at the Interscience Conference on Antimicrobial Agents and Chemotherapy, New Orleans, 1980) with the exception of the missing exanthema. She used tampons regularly and changed them frequently in the daytime, but used one tampon at night. This habit seems to be quite normal. During the previous period she had a one-day illness with high fever; and pain in the body and joints on the last day of the bleeding, but this illness cleared up spontaneously. This accords with the description of recurrence of toxic shock syndrome which occurs in about 30% of the patients.

Staph aureus of phage-group I was initially reported to be associated with toxic shock syndrome,[3] but this has not been substantiated from the United States. *E coli* is a known toxin producer. Whether finding *E coli* together with *Staph aureus* is important in the toxic shock syndrome has not been discussed.

This case, and the other reports from Sweden, show that the toxic shock syndrome is not an American problem only, but can occur in other parts of the world or at least in Scandinavia.

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Methylene blue is dangerous

SIR,—I was surprised to read the suggestion in "Any Questions" (11 October, p 981) that the intrathecal administration of methylene blue could be carried out to establish the occurrence of cerebrospinal fluid rhinorrhea.

Evans and Keegan[1] described 14 cases of neurotoxic adverse effects of intrathecal methylene blue; neurological sequelae included paraplegia, radiculopathy, cauda equina syndrome, encephalopathy, optic neuritis, and meningeal irritation. They concluded that methylene blue should not be administrated intrathecally. Schultz and Schwartz[2] described a patient who suffered extensive damage to the spinal roots and spinal cord following intrathecal methylene blue and reported an additional three cases.

The most recent report of neurological sequelae following this procedure is that of Sharr *et al*,[3] who describe a 59-year-old man who was given intrathecal methylene blue in an attempt to locate the source of cerebrospinal rhinorrhoea. He developed a progressive paraparesis with urinary retention, which progressed over 3½ years after the intrathecal