less than their median lethal dose. Comparative figures from Toronto show that at the Ontario Trauma Centre, which has an organised trauma system, only 6% of patients die with an injury severity score less than their median lethal dose.[3] The equivalent figure for other Toronto hospitals of a similar standard but lacking an organised trauma system was 53%.

I also examined the problems entailed in transferring patients with multiple injuries from outlying hospitals to the Royal Infirmary. In this group half were mismanaged in the outlying hospital to an extent that might have contributed to their death.

About 350 patients with multiple injuries are admitted each year to the Royal Infirmary, but despite the skill that should accompany this workload up to 70% of the hospital deaths after a road traffic accident seem to be avoidable. The reasons for this are twofold. Firstly, trauma has a low priority within the medical profession to the extent that decision making and treatment are frequently undertaken by junior staff with little experience. Secondly, poor communication exists among the various surgical disciplines, ensuring poor management of patients. The facilities in the hospital for dealing with those with multiple injuries are dreadful. The lack of high dependency and intensive care beds means that patients are usually managed in ordinary wards and if ventilation is required it is usually delayed. The ideal situation is the creation of trauma centres as suggested by the recent report, but until these are established we need to rethink our approach to trauma and to expand existing facilities if my unpalatable statistics for the Royal Infirmary are to be improved.

C M COURT-BROWN

University Department of Orthopaedic Surgery,
Clinical Research Unit,
Princess Margaret Rose Orthopaedic Hospital,
Edinburgh EH10 7ED

1 Yates DW. Action for accident victims. *Br Med J* 1988;**297**:1419-20. (10 December.)
2 Bull JP. The injury severity score of road traffic casualties in relation to mortality, time of death, hospital treatment time and disability. *Accid Anal Prev* 1975;7:249-55.
3 Wright CS, McMurtry RY, Pickhard J. A postmortem review of trauma mortalities—comparative study. *J Trauma* 1984;**24**: 67-8.

SIR,—In his editorial on the report of the Royal College of Surgeons on the management of patients with major injuries Mr D W Yates correctly emphasised the importance of professional enthusiasm in treating such patients successfully.[1] This is particularly true in district general hospitals because patients with life threatening injuries will continue to present there, many of whom will simply not be suitable for transfer to another hospital. I was recently concerned with managing a 19 year old man who developed a pneumothorax and cardiac tamponade after having been stabbed in the chest. He died as a direct result of our attempts to transfer him (on expert advice) to the local specialist centre and the delay that this entailed.

Cardiac tamponade occurs when a collection of fluid in the pericardial space leads to a rise in intrapericardial pressure and reduced cardiac filling. When the intrapericardial pressure rises above a critical value cardiac output falls to virtually zero[2]; the patient may seem stable while the intrapericardial pressure steadily rises but suddenly dies when the critical pressure is reached. Needle pericardiocentesis is unlikely to be helpful,[3] and urgent thoracotomy is the only effective means of treatment. Those who argue in favour of transferring such patients to the local specialist centre claim that it is wrong to carry out any cardiac surgery in a hospital where there are no facilities for cardiopulmonary bypass operations and no specialist cardiac surgeons. Clearly, from the above case, however, the condition of these patients is inherently unstable so that to transfer them elsewhere is inappropriate.[4] Furthermore, even in specialist centres the mortality among patients with complex cardiac injuries (the group for whom cardiopulmonary bypass operations may be required) is about 80%.[5]

If our patient had been managed in a specialist trauma centre in the first instance he would almost certainly have survived,[6] but the delay entailed in arranging interhospital transfer proved to be fatal. Therefore, though I welcome the general change in attitude recommended in the Royal College of Surgeons' report, I cannot agree with its recommendation that all patients presenting to a district general hospital with life threatening injuries should be transferred to a trauma centre.

A D SCOTT

St Bartholomew's Hospital,
London EC1A 7BA

1 Yates DW. Action for accident victims. *Br Med J* 1988;**297**: 1419-20. (3 December.)
2 Islam JP. Sixty penetrating wounds of the heart: clinical and experimental observations. *Surgery* 1959;**45**:696.
3 Ivatury RR, Shah PM, Ito K, *et al*. Emergency room thoracotomy for the resuscitation of patients with "fatal" penetrating injuries of the heart. *Ann Thorac Surg* 1985;**32**:377-85.
4 Taggart DP, Reece IJ. Penetrating cardiac injuries. *Br Med J* 1987;**294**:1630-1.
5 Campbell T, McCarthy M, Yaw P, *et al*. Penetrating cardiac injury: can we improve survival? *Indiana Med* 1987;**80**:31-7.
6 West JG, Trunkey DD, Lim RC. Systems of trauma care. A study of two countries. *Arch Surg* 1979;**114**:455-60.

SIR,—I hope that the recent recommendations of the Royal College of Surgeons working party will be discussed widely by profession and public, so that mismanagement of major injuries will no longer result in 5000 unnecessary deaths each year.[1]

The postwar pandemic of road traffic accidents was recognised in the 1950s and positive suggestions for improving care were made long ago by the Platt and accident services review committees of the BMA,[2] the first international symposium on immediate care in north Yorkshire, and the Medical Commission on Accident Prevention.[3] The apparent inability of government departments to perceive the gravity of the situation, plus financial restraints, has caused difficulties in the evolution of accident and emergency departments, a career structure for their consultants, and improved standards of equipment and care within the ambulance services.

Rehabilitation begins at the roadside, and advanced medical aid is required from the very beginning of the "golden hour." Since 1977 the British Association of Immediate Care Schemes has existed to coordinate efforts of volunteer doctors working as a team with the statutory services at scenes of sudden accident or illness. All this work is charitable, no payments are received from the National Health Service for attendance at road traffic accidents, and at present 2000 such volunteer doctors cover one third of the United Kingdom. BASICS doctors were at the recent Hungerford, Kings Cross, and motorway fog disasters.

The need for such medical care can now no longer be ignored by the government. Helicopters would be useful adjuncts to the teamwork but they do have many major limitations. Patients will still need stabilising before evacuation. BASICS is attempting to fill this "therapeutic vacuum," its member schemes and its essential work relying solely on charitable donations. Two thirds of the United Kingdom remain without cover from immediate care schemes, and many more volunteer doctors are invited to join us. Surely now is the time for parliament to vote us adequate funding towards this national need?

KENNETH C EASTON

Catterick Village,
Richmond, North Yorkshire DL10 7LN

1 Yates DW. Action for accident victims. *Br Med J* 1988;**297**:1419. (3 December.)
2 Accident Services Review Committee. *Report of a working party*. London: BMA, 1970.
3 Ministry of Health. *Accident and emergency services*. London: HMSO, 1962. (Platt report.)

## A surgical thought on disaster relief

SIR,—I found Mr John Patrick's stimulating article[1] a delight to read, having recently had the experience of commanding a Western style British military hospital in a Third World country during a disaster relief operation. This followed the Nepal earthquake on 21 August 1988, when we treated around 900 victims, over 450 on the first day. With our military back up and lines of communication all of Mr Patrick's criteria for success were met, and our results will be published elsewhere.

The surgical facilities for major disaster relief already exist in the form of containerised airportable hospitals, complete with generators and operating theatres, in the medical corps of many armies, including the British army. In the military environment expert and specialist staff are easily plucked from their base hospitals and dispatched at extremely short notice. Additionally, their frequent training in mass casualty handling is just as applicable to civilian as to military disasters.

Regrettably, there is more to disaster relief than medical manpower and hardware, and the politics of natural catastrophes in the Third World are confusing and occasionally shocking. Through our military and diplomatic channels we were able virtually to impose a highly successful relief operation on the shattered local community. A much needed French orthopaedic field surgical team waited in vain for three weeks in Paris for diplomatic clearance, which was in the end not forthcoming. Sadly, relief operations are normally invited by host governments and cannot be imposed by outside agencies. Many Third World régimes totter precariously on a political knife edge, and the overt inability of central government to cope with disaster relief on its own is viewed by those in power as a recipe for potential collapse. This, coupled with innate corruption, bureaucratic red tape, and the prevalent attitudes about life and death itself, can make the whole business of disaster relief in Third World countries a nightmare to run.

Until host governments overcome these characteristics it is unlikely that the developed nations' surgical skill will be available (on time) to disaster victims in the Third World. Politics and surgery are here inextricably intertwined. And, although airportable military hospitals may not have the political respectability of other aid agencies, they probably offer the best option at present.

P J GUY

British Military Hospital,
Hanover, West Germany

1 Patrick J. A surgical thought on disaster relief. *Br Med J* 1988;**297**:1349. (19 November.)

## DF-2 infection

SIR,—We agree with Drs Mark McCarthy and Alimuddin Zumla that DF-2 infection must be more common than the number of cases reported to the Public Health Laboratory Service Communicable Disease Surveillance Centre.[1] Recently we identified as DF-2 strains four organisms from blood cultures referred to us as possible campylobacters and isolated the organism from a dog bite wound. All patients but one had a history of close contact with dogs. Consequently we investigated the oral flora of cats and dogs attending veterinary

surgeries and have reported our early findings.[2] We extended our study to include animals for slaughter. As Drs McCarthy and Zumla refer to the organism being difficult to culture it is appropriate to present our methods, which by using a selective medium make the isolation and recognition of DF-2 organisms comparatively easy.

Cotton wool tipped swabs of incisor teeth and gingival margins were placed in Amies charcoal based transport medium for delivery to the laboratory, where they were cultured using brain-heart infusion agar (Difco) containing horse blood 5%, cysteine hydrochloride 0·5 g/l, kanamycin 25 mg/l, and vancomycin 1 mg/l. Plates were incubated in 95% air and 5% carbon dioxide in 95% relative humidity at 37°C and examined after three and five days. Under these conditions DF-2 grew after three days as smooth grey convex, circular colonies about 2-3 mm in diameter. Gram stained smears showed pleomorphic Gram negative bacilli with tapering ends, filamentous organisms being commonly seen. The identity of suspected isolates was confirmed using methods similar to those of Weaver *et al*[3] with the addition of rapid enzyme tests. In addition to organisms that conformed to previous descriptions of DF-2 others were isolated that fermented glucose, maltose, lactose, sucrose, and usually raffinose and inulin. They are thus similar to the DF-2 like strains reported by Weaver *et al*.[4]

Altogether 469 swabs were collected from dogs, cats, and other animals; the table shows the results.

*Isolation of DF-2 and DF-2 like organisms from oral swabs of domestic animals. Values are numbers (percentages)*

| | Total | DF-2 isolated | DF-2 like organisms isolated |
|---|---|---|---|
| Dogs | 180 | 44 (24) | 20 (11) |
| Cats | 249 | 42 (17) | 19 (8) |
| Pigs | 13 | | |
| Sheep | 12 | 3 (25) | |
| Cattle | 15 | 5 (33) | |

The isolation rate was higher than in the original survey reported by Bailie *et al*, who found the organism in the oral and nasal fluids of four out of 50 dogs.[5] Our findings support the view that domestic pets are the likely source of infection in humans, and it will be seen that the organisms are easily recovered from cats. Although there has been only one report of infection after a cat bite,[6] five DF-2 eye infections have been reported, of which three were associated with corneal cat scratch injuries.[7]

We have been unable to isolate DF-2 organisms from human oral flora (0/40 adults) using the same selective medium. This medium proved ideal, however, for the isolation of *Capnocytophaga* spp, which have recently been associated with infections in both immunocompromised and immunocompetent subjects.[8]

A J WESTWELL
KEVIN KERR
M B SPENCER
D N HUTCHINSON

Public Health Laboratory,
Preston PR1 6PS

1 McCarthy M, Zumla A. DF-2 infection. *Br Med J* 1988;**297**: 1355-6. (26 November.)
2 Westwell AJ, Spencer MT, Kerr KG. DF-2 bacteremia following cat bites. *Am J Med* 1987;**83**:1170.
3 Weaver RE, Hollis DG, Bottone EJ. Gram-negative fermentative bacteria and Francisella tularensis. In: Lennette EH, Balows A, Hausler WJ, Shadomy HJ, eds. *Manual of clinical microbiology*. Washington, DC: American Society of Microbiology, 1985:309-29.
4 Weaver RE, Fanning GR, Hollis DG, Brenner DJ. Classification of group DF-2: a cause of septicaemia following dog bite. In: Leclerc H, ed. *Les bacilles à gram négatif d'interêt médical et en santé publique: taxonomie-identification-applications*. Paris: INSERM Publications, 1983;**114**:351.
5 Bailie WE, Stowe EC, Schmitt AM. Aerobic bacterial flora of oral and nasal fluids of canines with reference to bacteria associated with bites. *J Clin Microbiol* 1978;7:223-31.
6 Carpenter PD, Heppner BT, Gnann JW. DF-2 bacteremia following cat bites—report of two cases. *Am J Med* 1987;**82**: 621-3.
7 Paton BG, Ormerod LD, Peppe J, Kenyon KR. Evidence for a feline reservoir for dysgonic fermenter 2 keratitis. *J Clin Microbiol* 1988;**26**:2439-40.
8 Warren JS, Allen SD. Clinical pathogenic and laboratory features of Capnocytophaga infections. *Am J Clin Pathol* 1986;**86**: 513-8.

## Luteinising hormone in polycystic ovary syndrome

SIR,—We wish to comment on the paper by Dr R Homburg and others on the influence of luteinising hormone concentrations on fertility treatment with luteinising hormone releasing hormone.[1]

Frequent sampling of luteinising hormone concentrations has shown that a single random blood sample analysis will give a result within 20% of the true mean value calculated over an eight hour period in only 30% of samples.[2] In the longer term mean luteinising hormone concentrations are also influenced by the hormonal milieu to which the pituitary is exposed. The raised values frequently found in the polycystic ovary syndrome tend to fall after an ovulatory cycle.[3] Because of these points we do not think that it is appropriate to draw the conclusions stated in this paper.

From the data given on basal luteinising hormone concentrations it may be concluded that a raised value before starting treatment is negatively associated with subsequent ovulation and conception. Raised values during treatment cannot, however, be thought of as the cause of the failure to ovulate or conceive unless the values are measured during the period of treatment.

When the authors measured luteinising hormone concentrations during treatment they chose to analyse a single arbitrarily selected data point on each patient which may not be representative of the mean luteinising hormone concentration. From these data raised luteinising hormone concentrations may have an adverse effect on ovulation and conception, given the limitations of a single sample per patient, but they do not show that raised luteinising hormone concentrations are a causal factor in early pregnancy loss. In the group in which luteinising hormone values were measured during the conception cycle the values in the group with early loss of pregnancy were the same as those in the group that proceeded to delivery. We do not wish to dismiss this attractive theory but would welcome more substantial evidence in its support.

ELAINE TURNER

Department of Gynaecology,
University of Newcastle,
Newcastle upon Tyne

MICHAEL C WHITE

Department of Medicine,
Royal Liverpool Hospital,
Liverpool L7 8XP

1 Homburg R, Armar N, Eshel A, Adams J, Jacobs HS. Influence of serum luteinising hormone concentrations on ovulation, conception, and early pregnancy loss in polycystic ovary syndrome. *Br Med J* 1988;**297**:1024-6. (22 October.)
2 Goldzieher JW, Dozier TS, Smith KD, *et al*. Improving the diagnostic reliability of rapidly fluctuating plasma hormone levels by optimised multiple-sampling techniques. *J Clin Endocrinol Metab* 1976;**43**:824-30.
3 Baird DT, Corker CS, Davidson DW, Hunter WM, Michie EA, Van Look PF. Pituitary-ovarian relationships in polycystic ovary syndrome. *J Clin Endocrinol Metab* 1977;**45**:798-809.

AUTHORS' REPLY,—As endocrinologists we cannot but accept the limitations of hormone measurements on isolated samples of biological material. None the less, the results in our paper that Drs Turner and White commented on may be more readily interpreted in the light of recent descriptions of the clinical presentation of patients with the polycystic ovary syndrome. For example, in a study of more than 500 patients with this condition 44% had a random serum luteinising hormone concentration that was raised, and infertility (but not hirsutism) was significantly more prevalent in these subjects than in those with normal luteinising hormone values (high *v* normal values: infertile 37% *v* 21%, $\chi^2=14{\cdot}1$, $p=0{\cdot}004$).[1]

Our paper was an attempt to determine the nature of this association of infertility with the hypersecretion of luteinising hormone. The results during treatment with pulsatile luteinising hormone releasing hormone, always reported as the mean of at least two measurements, were consistent with earlier studies both in experimental animals and in modern programmes of assisted fertility. We suggested that the hypersecretion of luteinising hormone exerts its adverse effect by causing premature completion of maturation of the oocyte contained in the follicle destined to ovulate. It is important, however, to recognise that luteinising hormone releasing hormone and its analogues may themselves have this effect.[2] The situation is therefore potentially extremely complex in that both the condition and treatments commonly used to correct it may share a common and adverse pathway. This is a point that may need emphasis again when the results of treatments designed to inhibit luteinising hormone secretion are evaluated.

Finally, on a technical matter raised by Drs Turner and White the serum luteinising hormone measurements during treatment, rather than being arbitrarily timed, were specifically scheduled for the phase of maximum follicular growth, when the follicle wall is presumably most readily penetrated by luteinising hormone.

R HOMBURG
H S JACOBS

Cobbold Laboratories,
University College and Middlesex School of Medicine,
London W1N 8AA

1 Conway GS, Honour J, Jacobs HS. Heterogeneity of the polycystic ovary syndrome: clinical, endocrine and ultrasound features in 556 patients. *Clin Endocrinol* (in press).
2 Deckel N, Sherizly I, Phillips DM, Nimrod A, Zilberstein M, Naor Z. Characterisation of the maturational changes induced by a GnRH analogue in the rat ovarian follicle. *J Reprod Fertil* 1985;75:461-6.

## Is there a genetic factor in flecainide toxicity?

SIR,—Dr J Beckman and others[1] reported the risk of flecainide toxicity in poor metabolisers of sparteine. We report a further cause of flecainide toxicity in a neonate with Wolff-Parkinson-White syndrome that we believe was related to the method of storage of his oral flecainide preparation.

A 2940 g term boy was transferred to our unit aged 4 hours after developing a supraventricular tachycardia of 300 beats/min during the second stage of labour. This recurred after birth and was resistant to treatment with vagal stimulation, direct current shock, and intramuscular digoxin. On transfer he was found to have a narrow QRS tachycardia (of 270 beats/min) with a 1:1 atrioventricular relation. Facial immersion in ice cold water restored sinus rhythm, and the electrocardiogram then showed delta waves and a short PR interval confirming Wolff-Parkinson-White syndrome. The tachycardia recurred and was successfully treated with intravenous flecainide (2·5 mg). He was discharged after four days and was given oral flecainide syrup prepared by the hospital pharmacy (20 mg twice daily, 5 mg/ml). A trough plasma concentration with this regimen before discharge was 901 µg/l (target range 400-1000 µg/l).

Nine days later he was reviewed on the ward. He was well but on examination had a slow irregular