be included—telephoning the patient before visiting, not carrying an identifiable medical bag, taking personal alarms, informing people of your whereabouts, using car telephones, working with the police and deputising services, and ensuring availability of numbered council estate maps. Local authorities should provide adequate lighting, naming, and security on estates.

Social security staff, local government workers, social workers, nurses, and health visitors have all had guidelines from their unions or professional bodies.[18] A few local medical committees such as Glasgow have taken initiatives, but the BMA and the Royal College of General Practitioners have done little, fearing that highlighting the problem will lead to copycat crimes. The General Medical Services Committee is to conduct a nationwide survey of the extent of the problem in general practice, but it is so urgent that the GMSC should immediately facilitate the development of national guidelines for local medical committees and general practices. Soon controversial contractual arrangements may well be introduced between family practitioner committees and doctors, and it would be better for the profession to make a voluntary system of controlling violence work rather than wait for the government to impose one. The GMSC must press the government to recognise that measures against violence require extra resources.[18]

Central to the problem in inner cities is the urgent need for improving premises. The reinstatement of the enhanced improvement grant and differential relaxation of cost rent limits to match building costs are vital, especially in London. The Department of Health should set up a proper national reporting system for violence in general practice and collect data from family practitioner committees, medical defence societies, deputising services, and the Criminal Injuries Compensation Board. Family practitioner committees with a high incidence of violence will need to press the department for more generous cash limiting of their ancillary staff budget—to encourage appointment of security staff, psychiatric social workers for staff counselling, and psychologists and extra nurses for assistance with drug and alcohol related problems. Family practitioner committees should consider appointing a facilitator in each area to oversee a policy for preventing violence, bringing together local authorities, the police, and general practitioners.

Practices take for granted preventing the spread of infection, but now we must spend time and money preventing the greater risk of violence.

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1 Savage R. Violence. *Br Med J* 1984;**289**:1618-9.
2 Cambrowicz S, Ford PGT, Winn JC, Goodman M, Dyer C. Assault on a GP. *Br Med J* 1987;**294**:616-8.
3 Walls F. Assault. *Br Med J* 1983;**286**:113-4.
4 Raghu K. Aggressive patients—what is the answer? *Br Med J* 1979;ii:1147.
5 Siriwardene SK. Doctor at risk. *The Physician* 1987;**6**(6):333.
6 Neville RG. Violent patients in general practice. *The Practitioner* 1986;**230**:1105-8.
7 Health and Safety Commission. Health Services Advisory Committee. *Violence to staff in the health services*. London: HMSO, 1987.
8 Health and Safety Commission. *Violence to staff: a basis for assessment and prevention*. London: HMSO, 1986.
9 Richardson P. "Frontline" GPs in danger of their lives in inner-city battle. *Pulse* 1987;**47**:20.
10 Rasheeduddin Khan. Violence and socioeconomic development. *Violence and its causes*. Paris: Unesco, 1975:167-85.
11 Brown R, Bute S, Ford P. *Social workers at risk: the prevention and management of violence*. London: Macmillan, 1986.
12 Kirby T. Crime in London being forced onto the streets. *Independent* 1988 Dec 16:4(cols 1-5).
13 Home Office. *Criminal statistics in England and Wales*. London: HMSO, 1987.
14 Anonymous. Assaults on doctors. *Br Med J* 1978;i:1229-30.
15 Short PW. The psychiatrically violent patient. *Br Med J* 1981;**282**:281-2.
16 Woolf PG. Subnormality and violence. *The Practitioner* 1982;**226**:503-4.
17 McKegoney N. Shadowland: general practitioners and the treatment of opiate abusing patients. *Br J Addict* 1988;**83**:373-86.
18 Department of Health and Social Security. *Violence to staff*. Report of DHSS advisory committee on violence to staff. London: HMSO, 1988.
19 Council of Family Practitioner Committees. Attacks and robberies involving doctors. *Family Practitioner Services* 1987;**14**:278-9.
20 Fricker J. So you think you're safe? *J R Coll Gen Pract* 1987;**37**:426-7.
21 Anonymous. Fear keeps GPs silent. *Pulse* 1988;**48**:12.
22 Anonymous. Compensation for attacks by patients. *Br Med J* 1985;**291**:1496.
23 Smith H. Shield staff from attack. *Medeconomics* 1985;**6**:81-2.
24 Poyner B, Warne C. *Preventing violence to staff*. London: HMSO, 1988.
25 Nichols J. Managing aggressive patients. *Pulse* 1986;**46**:36-46.
26 Stott P. Funding continuing medical education. *Medical Monitor* 1988 Dec 2:15.

Chancroid

Epidemics in some developing countries but still rare in Britain

Chancroid is a sexually transmitted disease causing painful destructive genital ulcers and enlarged inguinal lymph nodes that in time may suppurate. It is caused by *Haemophilus ducreyi*, a fastidious bacterium not easily cultured except in experienced hands. The disease is possibly the leading cause of genital ulceration in developing countries.[1-4] In the developed world the main cause is herpes simplex,[5] and chancroid is rarely seen or diagnosed.

Recently, however, outbreaks of chancroid have been seen in Greenland,[6] Rotterdam,[7] and various parts of the United States.[8-11] The epidemiology is similar in all these outbreaks: the cases mostly occur among poor, heterosexual people and often result from contact with prostitutes. In the United States many of the men were migrant workers. The association with prostitution has also been described in Nairobi, where three fifths of men with chancroid acquired it from prostitutes.[12]

Prostitutes are probably a source of infection more for economic than physical reasons. When facilities for diagnosing and treating genital complaints are cheap and freely available patients with genital ulceration will seek help and treatment. When the population is poor and facilities are limited or expensive patients may delay in seeking medical advice. This may apply particularly to poor prostitutes who rely on sexual activity for their subsistence.

In Britain chancroid is exceptionally rare: in 1965 only 74 cases in men and five in women were reported from clinics for sexually transmitted diseases. This represented 0·001% of all cases attending clinics, and the number seen each year since then has remained virtually static. There have always been many more cases among men than women, but in recent years the gap has narrowed: from 1965 to 1969 only 14 of the 362 (4%) patients were women compared with 114 of the 425 (27%) seen between 1981 and 1985. This may reflect an increased number of prostitutes.

In the early 1980s workers in Sheffield developed a modified culture medium said to be superior to the media used previously for culturing *H ducreyi*.[13-15] Using this medium they cultured an organism said to be *H ducreyi* from over a quarter of patients with genital ulcers as well as from several people without symptoms presenting to the local clinic for sexually transmitted diseases.[14,16] These studies are contrary to those from other Western cities, including Liverpool,[17] Manchester,[18] Winnipeg,[19] and Antwerp,[20] where

H ducreyi was isolated only rarely with conventional media and always from people with genital ulcers, which were usually acquired abroad. The Sheffield medium has been tested in the tropics, and its lack of sensitivity suggests that it is detecting organisms other than *H ducreyi*.[20]

The Sheffield results thus seem to have been an artefact, and chancroid remains a very rare imported condition in Britain. As long as British facilities to treat genital ulcers remain free and easily accessible this state of affairs is likely to continue.

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1 Duncan MO, Bilgeri YR, Fehler HG, Ballard RC. The diagnosis of sexually acquired genital ulcerations in black patients in Johannesburg. *South Africa Journal of Sexually Transmitted Diseases* 1981;1:20-3.
2 Nsanze H, Fast M, D'Costa LJ, Tukei P, Curran J, Ronald AR. Genital ulcers in Kenya: a clinical and laboratory study. *British Journal of Venereal Disease* 1981;57:378-81.
3 Meheus A, Van Dyck E, Ursi JP, Ballard RC, Piot P. Etiology of genital ulcerations in Swaziland. *Sex Trans Dis* 1983;10:33-5.
4 Taylor DN, Duangmani C, Suvongse C, *et al*. The role of Hemophilus ducreyi in penile ulcers in Bangkok, Thailand. *Sex Trans Dis* 1984;11:148-51.
5 Adler MW. *ABC of sexually transmitted diseases*. London: British Medical Association, 1984:19-20.
6 Lykke-Olesen L, Larsen L, Pedersen TG, Gaarslev K. Epidemic of chancroid in Greenland 1977-1978. *Lancet* 1979;i:654-5.
7 Nayyer KC, Stolz E, Michel ME. Rising incidence of chancroid in Rotterdam. Epidemiological, clinical, diagnostic, and therapeutic aspects. *British Journal of Venereal Disease* 1979;55:439-41.
8 Blackmore CA, Limpakarnianarat K, Rigau-Perez JG, *et al*. An outbreak of chancroid in Orange County, California: descriptive epidemiology and disease control measures. *J Infect Dis* 1985;151:840-4.
9 Anonymous. Chancroid-Massachusetts. *MMWR* 1985;34:711-8.
10 Becker TM, DeWitt W, Van Dusen G. Hemophilus ducreyi infection in South Florida. A rare disease on the rise? *South Med J* 1987;80:182-4.
11 Schmid GP, Sanders LL, Blount JH, Alexander ER. Chancroid in the United States: re-establishment of an old disease. *JAMA* 1987;258:3265-8.
12 Plummer FA, D'Costa LJ, Nsanze H, Dylewski J, Karasira P, Ronald AR. Epidemiology of chancroid and Haemophilus ducreyi in Nairobi, Kenya. *Lancet* 1983;ii:1293-5.
13 Hafiz S, Kinghorn GR, McEntegart MG. Chancroid in Sheffield: a report of 22 cases diagnosed by isolating Haemophilus ducreyi in a modified medium. *British Journal of Venereal Disease* 1981;57:382-6.
14 Kinghorn GR, Hafiz S, McEntegart MG. Pathogenic microbial flora of genital ulcers in Sheffield with particular reference to herpes simplex virus and Hemophilus ducreyi. *British Journal of Venereal Disease* 1982;58:377-80.
15 Hafiz S, McEntegart MG, Kinghorn GR. Sheffield medium for cultivation of Haemophilus ducreyi. *British Journal of Venereal Disease* 1984;60:196-8.
16 McEntegart MG, Hafiz S, Kinghorn GR. Haemophilus ducreyi infections: time for reappraisal. *Journal of Hygiene* 1982;89:467-8.
17 Arya OP, Cupit P, Dundon MB, Bartzokas CA. Chancroid in Liverpool. *Genitourin Med* 1988;64:66.
18 Mallard RH, Macaulay ME, Riodeu T, Chowdhury FH, Chandiok S, Bhattacharyya MN. Haemophilus ducreyi infection in Manchester. *Lancet* 1983;ii:283.
19 Diaz-Mitoma F, Benningen G, Slutchuk M, Ronald AR, Branham RC. Etiology of nonvesicular genital ulcers in Winnipeg. *Sex Trans Dis* 1987;14:33-6.
20 Piot P, Slootmans L, Nsanze H, Ronald AR. Isolating Haemophilus ducreyi. *Lancet* 1983;ii:909-10.

Causes of the haemolytic uraemic syndrome

It might be verocytotoxin produced by Escherichia coli

The haemolytic uraemic syndrome is the commonest cause of acute renal failure in children in Britain and is increasingly recognised in adults.[1,2] Sporadic cases and small epidemics occur,[3,4] commonly in the summer months. The syndrome is characterised by a microangiopathic haemolytic anaemia, thrombocytopenia, and renal failure.[5] Most affected children (up to 90%) have the "classic" syndrome with prodromal diarrhoea that is usually bloody. Many causes of the syndrome, mainly infectious agents,[5-8] have been proposed, but recently a strong association has been found between the haemolytic uraemic syndrome and enteric infection with verocytotoxin-producing *Escherichia coli*.[9,10] These organisms, also known as enterohaemorrhagic *E coli*, are associated with clinical conditions ranging from self limiting diarrhoea to bloody diarrhoea with haemorrhagic colitis and the haemolytic uraemic syndrome.[8,11] Verocytotoxin (Shiga-like toxin) is also produced by *Shigella dysenteriae* type 1.[12]

The toxin consists of a biologically active subunit A linked to B subunits,[13] which bind to specific receptors.[14] Once attached to the cell surface subunit A enters the cell and inhibits protein synthesis by inactivating 60s ribosomal subunits, which leads to cell death.[15]

An infective cause for the haemolytic uraemic syndrome has long been suspected because of its seasonal distribution, the occurrence of small epidemics and person to person transmission, and its common association with prodromal gastrointestinal illness. Much of the pathology of the syndrome suggests that it is associated with a toxin,[8,16] and evidence to implicate verocytotoxin has accumulated. Infection with verocytotoxin-producing *E coli* was found in almost 90% of patients with the classic haemolytic uraemic syndrome studied in Canada (N Ish-Shalom *et al*, international symposium and workshop on VTEC [verocytotoxin-producing *E coli*] infections, Toronto, 1987), almost three fifths of those studied in the United States,[17] and a third of those studied in Britain.[18] The two pathogens most consistently associated with the syndrome—some serotypes of *E coli* and *Sh dysenteriae*—both produce high concentrations of verocytotoxin.[19] Verocytotoxin is found in faeces, and patients with the haemolytic uraemic syndrome may develop specific antibodies that neutralise the toxin.[10] Verocytotoxin has not been detected in patients' blood or tissues, but this is true of many diseases associated with toxins.

In vitro studies support the hypothesis that verocytotoxin may be important in the haemolytic uraemic syndrome. The causative agent must be able to damage endothelial cells,[5-7] and verocytotoxin has now been shown to be cytopathic to cultured endothelial cells.[20,21] It also causes the release of endothelial cell factor VIII (J Kavi *et al*, international workshop on VTEC infections, Toronto, 1987). These findings might explain the increase in large multimetric forms of factor VIII that is found in the plasma of patients with the acute phase of the haemolytic uraemic syndrome and returns to normal on clinical recovery.[22] These large forms of factor VIII promote platelet aggregation, thrombotic lesions, and thrombocytopenia. A potent platelet aggregating activity, which is independent of factor VIII, is also known to develop on incubating verocytotoxin with normal plasma.[23] Although the red cell fragmentation and haemolysis seen in the haemolytic uraemic syndrome are widely believed to be caused by mechanical injury, verocytotoxin might be the cause because red cell membranes have recently been found to contain specific verocytotoxin receptors.[14] Animal studies also suggest that verocytotoxin might be important in the haemolytic uraemic syndrome: verocytotoxin given intravenously in rabbits produces gross and ultrastructural changes similar to those seen in the haemolytic uraemic syndrome (S E Richardson *et al*, international workshop on VTEC infections, Toronto, 1987).

These advances raise as many questions as they answer, and a clear model to explain how verocytotoxin is associated with the haemolytic uraemic syndrome is still awaited. But